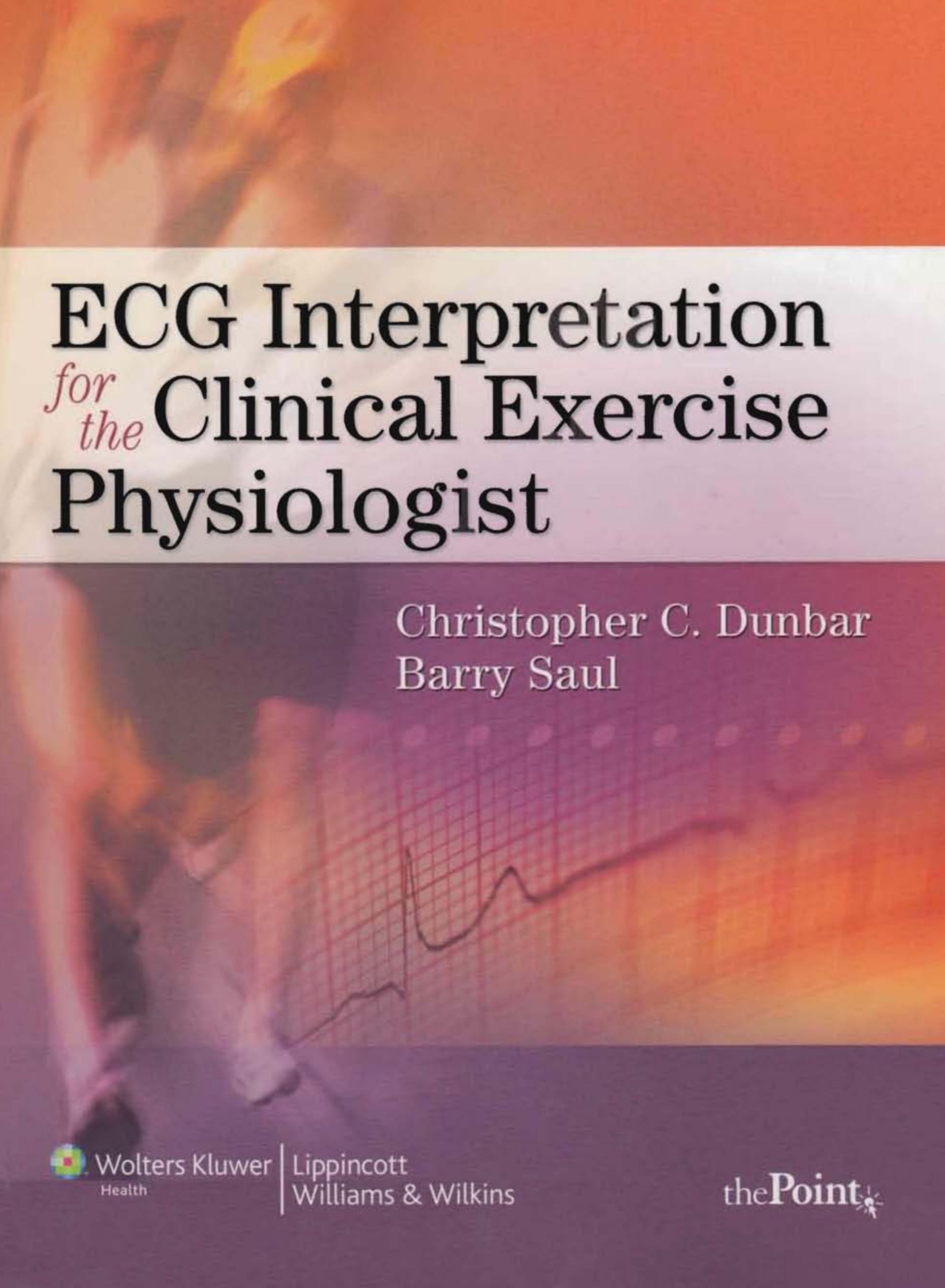

ECG Interpretation *for the* Clinical Exercise Physiologist

Christopher C. Dunbar
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ECG
Interpretation for the Clinical Exercise Physiologist

Christopher Dunbar, PhD, MPH

Professor

Center for Exercise and Sports Medicine

Brooklyn College

Brooklyn, New York

Barry Saul, MD

Department of Cardiology

New York Methodist Hospital

Brooklyn, New York

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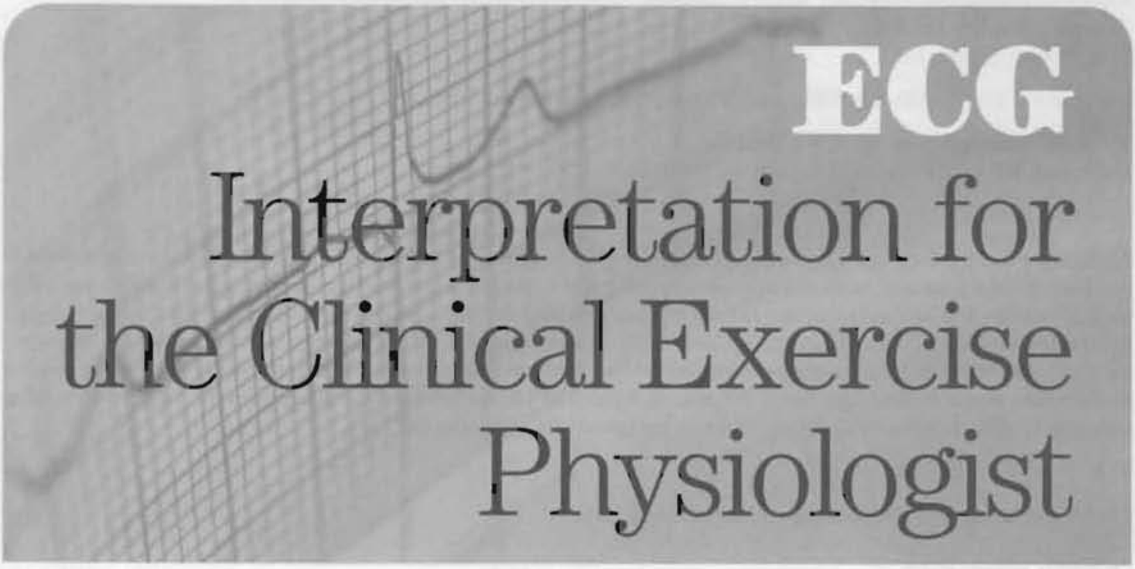

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Christopher Dunbar, PhD, MPH
Professor
Center for Exercise and Sports Medicine
Brooklyn College
Brooklyn, New York

Barry Saul, MD
Department of Cardiology
New York Methodist Hospital
Brooklyn, New York

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RRS0810

For Vasiliki, Andrew, Peter, Ellen, Karen, Michael, and Lauren.

Foreword

As a practitioner in the field of clinical exercise physiology I feel that the field has been missing a practical transition—one that presents sound and easy-to-follow advice for ECG interpretation and analysis and also provides a thorough and accurate depiction of the physiological basis behind the analysis. It is evident from the beginning of *ECG Interpretation for the Clinical Exercise Physiologist* that this book provides a unique approach to ECG interpretation. The text is written from the perspective of both a registered clinical exercise physiologist and a cardiologist, which is important because physiologists will often work with a team of cardiologists to determine testing outcomes and exercise prescriptions for cardiac patients: *ECG Interpretation for the Clinical Exercise Physiologist* presents an amalgamation of both perspectives.

The authors have, combined, more than 60 years of clinical experience, and this is evident throughout each chapter. This textbook's uniqueness lies in that it specifically targets those interested in clinical exercise physiology as a career, providing the reader with both an appropriate depth for graduate-level ECG interpretation as well as presenting "real life" examples of situations that are common in the field. It provides the reader with explicit examples at the beginning of the chapter (clinical vignettes) that precede the fundamental principles within the chapter. Subsequently, the authors complete the chapter by finishing the previously proposed scenario and providing a very effective and practical application toward their learning outcomes.

Most ECG books seem to be written for nonclassroom use. I do not recall ever seeing a book written by someone who actually teaches in the traditional setting. The approach in this book was largely distilled from experiences teaching ECG in the classroom to exercise science students, and this has resulted in a somewhat different approach than other books I am familiar with. The capacity of *ECG Interpretation for the Clinical Exercise Physiologist*, however, extends well beyond the classroom. It proves to be an easy and accurate reference tool in both clinical and rehabilitation settings. Both clinical exercise physiologists and aspiring students will welcome a text such as this: one that provides them with not only an extensive list of arrhythmias but also potential causal factors associated with each interpretation. This makes the text particularly relevant to those either seeking employment in or who are currently employed in clinical stress testing and cardiac rehabilitation.

Jason Siegler, PhD, ATC
Department of Sport, Health & Exercise Science
University of Hull
United Kingdom

Preface

With the continuing evolution of health care, exercise specialists/physiologists working in stress testing and cardiac rehabilitation have been asked to take on greater responsibilities, and they need to be confident in their ECG skills. A book that is up-to-date and comprehensive yet readable for the novice is necessary; it is also necessary that it includes information and examples specific to the needs of stress testing and cardiac rehabilitation. The ideal text would cover material at a suitable depth and breadth for students to prepare for the American College of Sports Medicine (ACSM) Exercise Specialist Certification and the ACSM's Clinical Exercise Physiologist Registry.

The need for an introductory text about ECG interpretation as described above became clear after years of teaching a lecture course in electrocardiography for exercise science students. Although bookstore shelves are already straining under the weight of an impressive array of books about ECG interpretation, we could not find a book that suited our needs.

ECG Interpretation for the Clinical Exercise Physiologist has been designed as an introductory textbook to be used in ECG courses taught to exercise science students and assumes no previous knowledge of the subject. It can also be used as a stand-alone resource for those who wish to learn about or hone ECG interpretation skills outside of the classroom environment (e.g., medical residents, nurses, physician assistants, etc.), or those reviewing ECG before professional tests such as the medical boards or advanced cardiac life support certification.

Organization

In this book we use a modular system and cover all major areas of ECG interpretation. Our favored approach teaches rhythm first; it is less intimidating and more engaging than jumping right into concepts of leads, infarct, and ischemia. After a chapter introducing basic concepts and measurements, the first half of the book is devoted to rhythm and atrioventricular blocks. This serves as an excellent lead-in to the second half, in which topics such as infarct, hypertrophy, axis, and conduction defects are covered. However, these topics *can* be discussed before rhythm by beginning with the first chapter and then moving to the second half of the book before returning to finish up with rhythm.

Cardiograms have been provided for review and practice at the end of each chapter, and an entire chapter is solely devoted to quiz and review. In the review chapter we introduce a simple yet effective system for reading ECGs wherein the interpreter moves from left to right (P wave to T wave). This technique minimizes memorization and provides an easily understood framework to make sure that nothing is missed. The review section is also an excellent tool for those who previously learned ECG reading and are reviewing for certification examinations.

The approach is very practical and at a level consistent with the ACSM's Knowledge, Skills, and Abilities (KSAs). Theory is provided to assist understanding but avoided when it would complicate the issue at hand. Unavoidably, certain things must be memorized; however, we try to emphasize the understanding of concepts wherever possible because we firmly believe this will result in better long-term retention of the material.

The figures are mostly derived from real patients' ECGs and have been honed over several years of use in teaching electrocardiography in an exercise science program. Because we have used this material in our programs with students from a variety of backgrounds, the text has also benefited from student feedback. The majority of these students were enrolled in a master's level program in exercise science and came from a variety of undergraduate backgrounds, including exercise science and other sciences (e.g., biology), as well as liberal arts such as English, sociology, and psychology. Some students had no clinical experience whatsoever, whereas others came from various health professions like physical therapy, nursing, occupational therapy, respiratory therapy, and medicine. Comments and feedback from these students were invaluable in refining the text and figures.

Features

Clinical information and case studies of particular utility for the exercise specialist and the registered clinical exercise physiologist are included in the clinical vignettes. These vignettes introduce a patient and ECG to the reader. The clinical vignette revisited at the end of the chapter discusses proper resolution of the issue or handling of the patient. Unique KSA icons are used to indicate sections that cover Knowledge, Skills, and Abilities (KSAs) for the exercise specialist and/or registered clinical exercise physiologist, as outlined in the *ACSM's Guidelines for Exercise Testing and Prescription 7e*. The icon is used to indicate that a topic is required knowledge for the ACSM exercise specialist and registered clinical exercise physiologist examinations. The quiz at the end of each chapter helps the student review concepts from that chapter.

Ancillaries

A comprehensive Web site link accompanies the book. Students are provided with fresh examples of strips (different from those in the text), which offer review and quizzes on basic measurements, rhythm, comprehensive 12-lead interpretation, and exercise testing. Instructors have access to answers to the in-text quizzes; a discussion and rationale of the appropriate answer is included. An image bank compiling all of the strips in the book and a PowerPoint lecture outline are also available.

ECG Interpretation for the Clinical Exercise Physiologist provides a sorely lacking ECG resource for the exercise specialist/physiologist and others involved in cardiac rehabilitation and stress testing. The information is pertinent, accessible, and up-to-date. Combined with the website materials, this text is a powerful tool for learning ECG interpretation.

We greatly acknowledge the support of Big Boss Deborah Rainaldi, Dr. Xia Fang, Jaclyn Apicello, Lee Tyler, Carmen Gonzalez, Juanita Anderson, all of our colleagues and patients, and the team at Lippincott Williams & Wilkins.

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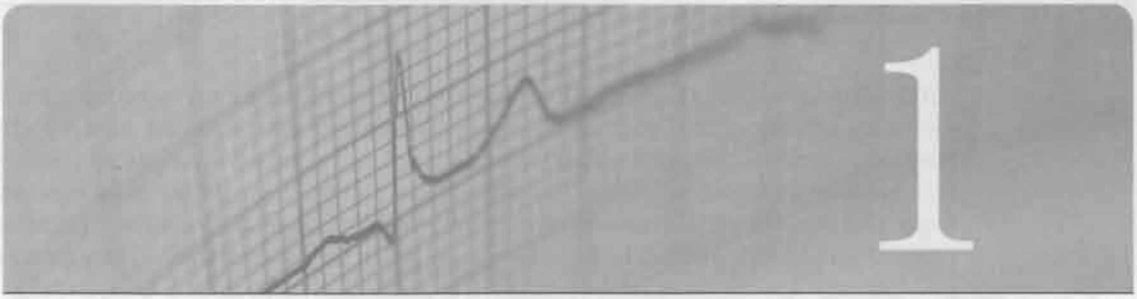


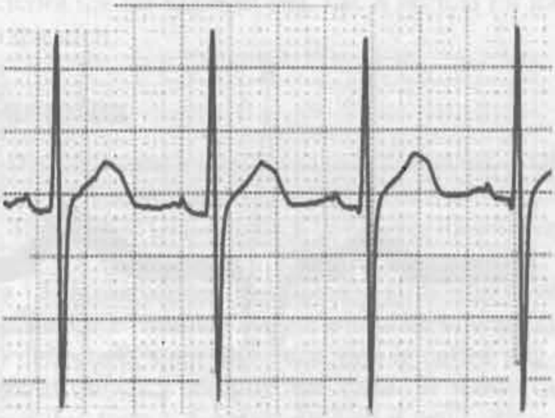
1

BASIC TERMINOLOGY AND MEASUREMENTS

P Wave
PR Segment and PR Interval
QRS Complex
T Wave
U Wave
ST Segment
ECG Paper
Calibration
Measurement of Intervals and Complexes
Measurement of Rate
Leads
Normal Ranges for Selected ECG Parameters

CLINICAL VIGNETTE

While working in the stress lab of a busy hospital, a clinical exercise physiologist receives a request for an exercise test on an inpatient. This patient is taking a medication called procainamide, which may prolong repolarization of the ventricles. The request specifies that the stress test should not be performed if the QT interval is "prolonged."



An electrocardiogram (ECG, or EKG, from the German *kardio*) is a recording of the electrical activity of the heart. As the recording electrodes are placed on the body surface, it forms a composite of the electrical activity of numerous cells and therefore appears quite different from the action potential tracings of individual cardiac cells. The purpose of this book is practical, to teach skills of ECG analysis; electrophysiological concepts will be discussed only when they facilitate ECG interpretation. However, as most of the basic principles of ECG interpretation are quite logical, it is more productive to think in terms of the basic underlying electrical events than to simply memorize criteria for the various abnormalities. A little understanding will go a long way toward learning, and perhaps more importantly, retaining the ability to successfully interpret an ECG.

KSA P Wave

Normally, the first electrical event of a cardiac cycle is the depolarization of the atria. This depolarization begins in the sinoatrial (SA) node and spreads through the atria from cell to cell. Since the atria are relatively small, thin walled chambers, the ECG will typically show a rather small waveform, which is termed the P wave **(Fig. 1.1)**. Beginning the ECG alphabet with the letter P has no clinical significance. It has been reported that P was chosen as the letter to describe the first wave by Willem Einthoven (the father of electrocardiography) in emulation of the style of the mathematician Rene Descartes, who often began his formulae with the letter P. The normal P wave should be <120 ms in duration and <0.25 mV in amplitude. How to measure the duration and amplitude of ECG events will be discussed shortly.

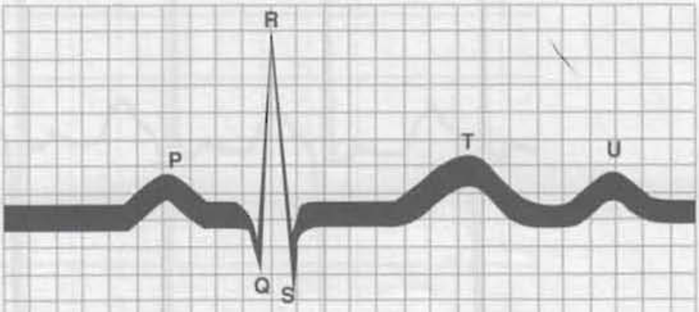


FIGURE 1.1 ECG waves.

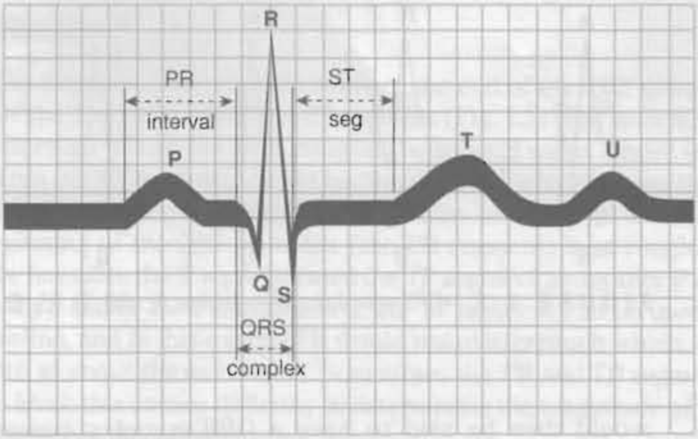


FIGURE 1.2 Waves, intervals, and segments.

PR Segment and PR Interval

A brief "pause," the PR segment, usually occurs after the P wave. In ECG terminology segments are sections intervening between waves. Following depolarization of the atria, a brief time is needed for atrial contraction and the subsequent completion of filling of the ventricles; thus, this "pause" should make intuitive sense. The PR segment represents the electrically quiet period between atrial and ventricular depolarization. The PR segment does not include any waves; it is the region after the P wave and before the QRS complex.

Intervals include both waves and segments, thus the PR interval is the region stretching from the beginning of the P wave to the beginning of the QRS complex, and it includes the PR segment **(Fig. 1.2)**. A normal PR interval is between 120 and 200 ms in duration.

QRS Complex

Normally the QRS complex follows the PR segment. A QRS complex may lack a Q, an S, or even an R wave, or have multiple R or S waves, yet it is always called a QRS complex. The normal QRS complex (whatever its components) should be <100 ms in duration.

Figure 1.3 shows various examples of QRS complexes (P and T waves are not shown). If the QRS complex begins with a downward (by convention also called negative) deflection, then this deflection is called a Q wave. Upward (positive) deflections in the QRS complex are called R waves. A deflection coming back toward baseline from below is called an S wave. Thus if the QRS complex consists of only a positive deflection that then returns to baseline (without ever being below baseline), it would be said that the QRS complex consists solely of an R wave. If the first deflection is negative followed by a positive deflection, it

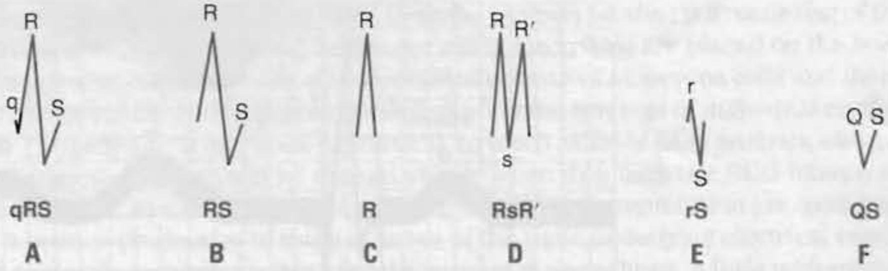


FIGURE 1.3 QRS complex morphologies. **A:** qRS. **B:** RS. **C:** R. **D:** RsR′. **E:** rS. **F:** QS.

would then be said to have a QRS complex consisting of Q and R waves. Sometimes, the QRS complex has a second R wave after an S wave; this is termed an R′ (stated as "R prime").

The size of the waves can also be described in relative terms. A QRS complex consisting of a small Q wave followed by a large R wave and a small S wave might be written as qRs. The same complex could be described on the phone to a colleague as a "small q, large R, small s." This terminology allows the reader/listener to form a relatively clear mental picture of the appearance of the QRS complex. Figure 1.3 illustrates the use of this terminology.

T Wave

The last major electrical event of the normal cardiac cycle is ventricular repolarization, which appears on the ECG as the T wave (Fig. 1.1). In contrast to the P wave, which often is symmetrical, the normal T wave typically is asymmetrical. Generally, T waves are also larger than P waves. In later sections we will see that the shape of the T wave can be of great clinical significance.

Repolarization of the atria is usually not seen on an ECG. The usual explanation for this is that it typically occurs at the same time as ventricular depolarization; therefore, the small T_a wave of atrial repolarization is thought to be obscured by the much larger QRS complex of ventricular depolarization. In fact this may not be the case. In abnormalities wherein P waves are dissociated from the QRS complex, T_a waves are still not commonly observed (although they theoretically should be).

U Wave

Sometimes a small complex known as the U wave follows the T wave (Fig. 1.1). The U wave is believed to represent the terminal stages of ventricular repolarization, possibly the repolarization of the Purkinje network. Often U waves are not present.

ST Segment

As implied by the name, the segment between the end of the QRS complex and the beginning of the T wave is called the ST segment (Fig. 1.2). The term ST segment is used even in cases where the QRS complex lacks an S wave. Under most normal circumstances the ST segment should be "isoelectric," which means that it should be on the baseline. Notice in Figure 1.2 that the line between the end of the P wave and the beginning of the QRS complex (the PR segment) is at roughly the same height as the line after the T wave (termed the TP segment as another P wave would appear shortly). Either the PR segment or the TP segment can be used as the isoelectric baseline, and in this example it would not matter much which is chosen as they are both at about the same level. Sometimes the PR and TP segments are not at the same level; this causes difficulty in determining the magnitude of parameters such as ST segment deviation.

ECG Paper

As shown in **Figure 1.4**, the paper that is normally used to record ECGs has a grid pattern consisting of thin lines every millimeter in both the horizontal and vertical planes, with thicker lines every 5 mm. On the horizontal, or X, axis, each of these 1-mm boxes represents 40 ms (0.04 sec) of elapsed time. Thus, five of these "small" (1 mm) boxes, or one "big" (5 mm) box is equivalent to 200 ms (0.20 sec). On the vertical, or Y axis, each 1-mm box represents 0.1 mV of voltage, resulting in 10 mm of upward deflection (ten small or two big boxes) being equivalent to 1 mV (10 × 0.1 mV). The preceding values assume standard calibration.

As previously stated, baseline refers to portions of a cardiogram where no net electrical activity is reflected, for example between the T wave of one cycle and the

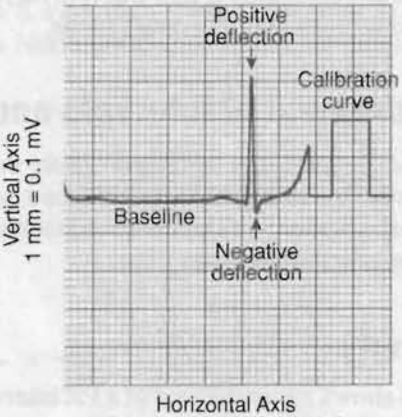
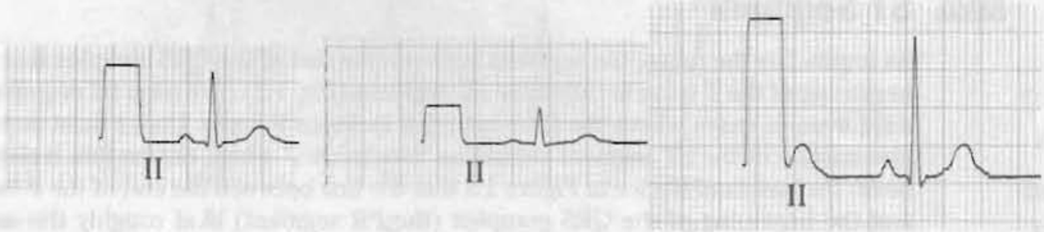


FIGURE 1.4 ECG paper. Each "thin" line equals 1 mm. Each "thick" line equals 5 mm. The calibration box is 10 mm tall (1.0 mV).



Standard Half 2×

FIGURE 1.5 Calibration.

P wave of the next (the TP interval). By convention upward deflections are referred to as *positive* and those moving downward are called *negative*.

Calibration

Virtually all ECG machines will give some indication of calibration. This may be in the form of a 1 mV (10 mm at standard calibration) box as shown in Figure 1.4 or by the printing of a phrase such as "1 cm = 1 mV" on the paper. Calibration should be checked on all ECGs, as most machines are capable of varying calibration. Very large complexes may run off the paper and require the calibration to be changed to half (1 mV = 5 mm); conversely very small ECG complexes can be enlarged for closer inspection by setting calibration to 2X (1 mV = 20 mm). The effect of varying calibration is shown in **Figure 1.5**.

Paper speed can also be varied. Standard paper speed is 25 mm/sec, resulting in each 1 mm box representing 40 ms (0.04 sec) of elapsed time on the X axis. Most machines also can be set to run at half (12.5 mm/sec) or double (50 mm/sec) speed. Under these conditions, 1 mm will represent 80 ms (0.08 sec) and 20 ms (0.02 sec), respectively, instead of the usual 40 ms.

Measurement of Intervals and Complexes

The grid of 1- and 5-mm boxes previously described can be used to quantify various ECG events. The measurements described below assume normal calibration (1 mV = 10 mm; paper speed = 25 mm/sec). Adjustments must be made if calibration is varied from these standard settings.

PR Interval

Figure 1.6 shows measurement of a PR interval. The PR interval begins at the beginning of the P wave and ends at the beginning of the QRS. The measurement is begun where the P wave leaves the baseline whether the initial deflection of the P wave is positive or negative (usually, as in this case, it is positive). Similarly measurement

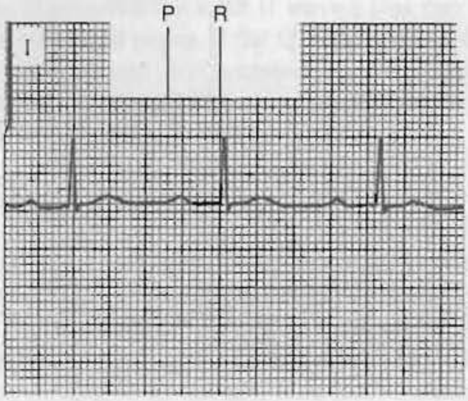


FIGURE 1.6 PR interval. The PR interval shown here is 280 ms (0.28 sec).

of the PR interval ends where the QRS complex begins, regardless of whether the QRS begins with a positive (R) or a negative (Q) wave. The vertical lines under the P and R in Figure 1.6 show where to begin and end measurement. A normal PR interval is between 120 and 200 ms (0.12 to 0.20 sec), this is equivalent to three to five of the small (40 ms) boxes on the ECG paper. Typically the PR interval is measured in lead II of the 12-lead ECG, although some authorities recommend measuring it in whichever lead shows the longest PR interval. The concept of leads will be briefly introduced later in this chapter and is dealt with in more detail in Chapter 7.

QRS Duration

Measurement of QRS duration **(Fig. 1.7)** begins wherever the initial deflection of the QRS begins, whether it is positive (R wave) or negative (Q wave). The end point for the QRS is the end of the S wave if an S wave is present or the end of the R wave if an S wave is not present. Normally the QRS duration should be <100 ms (2.5 small boxes) in all leads.

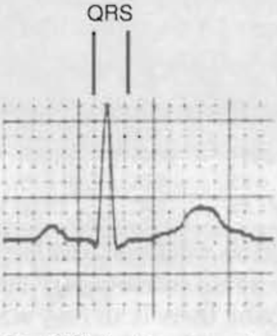


FIGURE 1.7 QRS duration. The QRS duration shown here is 80 ms (2 small boxes).

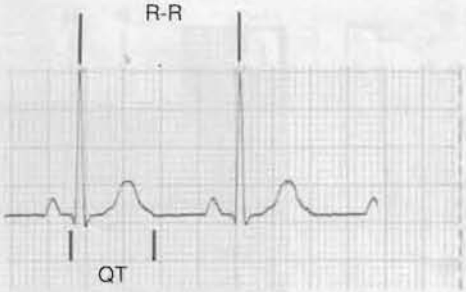


FIGURE 1.8 QT and R-R intervals. The figure shows a QT interval of 0.44 sec (440 ms) and an R-R interval of 0.88 sec (880 ms).

QT Interval

The QT interval **(Fig. 1.8)** is measured from the beginning of the QRS complex to the end of the T wave (and is called a QT interval even if the QRS complex does not begin with a Q wave). The length of the QT interval will normally vary with heart rate, so one normal value cannot be described. Standard tables for rate-specific values can be consulted, or the QT interval can be "corrected" for heart rate. A rate-corrected QT interval is abbreviated QTc. Bazett's formula, shown below, is often used to calculate a QTc. Although this formula is commonly applied to both sexes, some authorities recommend slightly different QT adjustments for males and females. The QT interval should be measured in whichever lead it is longest.

It is often difficult to accurately measure the QT interval, as determination of the precise ending of the T wave is often difficult. This is particularly true if a U wave merges with the T wave. In such cases an accurate QT interval cannot be determined.

BAZETT'S FORMULA

$$QTc = \frac{QT}{\sqrt{R - R}}$$

where QT is the QT interval in sec and R-R is the time from one R wave to the next R wave in seconds. Note: with this formula the values must be entered in seconds, not milliseconds.

The QTc should be <440 ms (0.44 sec). Most modern ECG machines will automatically calculate a rate corrected QT interval. If an automatic correction is not available or is in doubt, there is an easy way to estimate if the QT is longer than normal. At normal rates the QT interval should normally be less than half of the R-R interval (the interval from one R wave to the next R wave); this can readily be estimated using ECG calipers or a scrap of paper. If the QT interval is half the R-R interval (as in this case) or more, a QTc should be calculated. For this example the QTc = $0.44 \div \sqrt{0.88}$ or 0.47 sec (470 ms). A normal QTc should be <0.44 sec (440 ms), so this QT interval is prolonged.

Measurement of Rate

One of the most basic (yet critical) measurements made on virtually all ECGs is the assessment of heart rate (HR). The normal range of resting HR is between 60 and 100 beats per minute (bpm). Most ECG machines will measure the average HR and print it on the cardiogram, so it might seem superfluous to be able to measure rates. Unfortunately, the rate measured by the machine can be inaccurate. Further, when HR is varying it is often necessary to know the rate at different time points on a single ECG; machines only provide the average rate. To cover the full range of possibilities, clinicians should know at least two and perhaps three ways to measure HR. In cases where the rate is irregular it is sometimes best to describe the lowest and highest rates as well as the average. For example, one might report that the rate was varying between 60 and 140 bpm, with an average rate of 90 bpm.

1,500 Method (Most Accurate, Slower, Requires Arithmetic)

The *1,500 method* of determining heart rate is often used when very accurate determinations are needed. A 6- or 10-sec strip can be run and the rate for each R-R interval determined and then averaged. As mentioned earlier, an R-R interval is the distance from an R wave to the following R wave. At normal calibration (paper speed of 25 mm/sec) the R-R interval in millimeters divided into the constant 1,500 yields the heart rate during that R-R interval. For example, if the R-R interval is 22 mm, the heart rate during that interval is 68.18 bpm (1,500 ÷ 22 = 68.18). If the R-R intervals are fairly consistent, one measurement can yield a good estimation of the average rate. If the R-R intervals vary, several R-R intervals can be measured and the average rate determined. For greatest accuracy every R-R interval on the strip is measured and the average rate determined. This method is illustrated in **Figure 1.9**. If R waves are not present another consistent QRS landmark, such as the point of the S wave, can be used. A variation of this method is to measure the R-R interval in seconds (not millimeters) and divide it into 60. An example is shown in **Figure 1.10**.

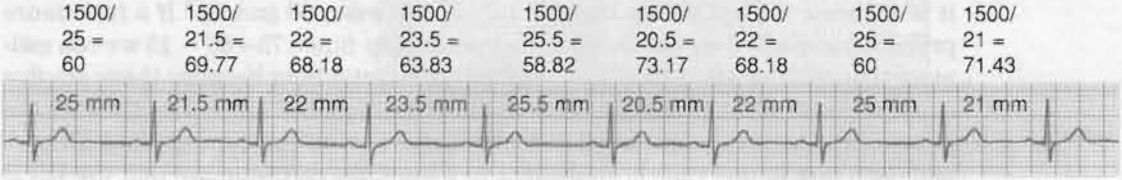


FIGURE 1.9 Heart rate 1,500 method. The average heart rate here is 65.93 bpm.

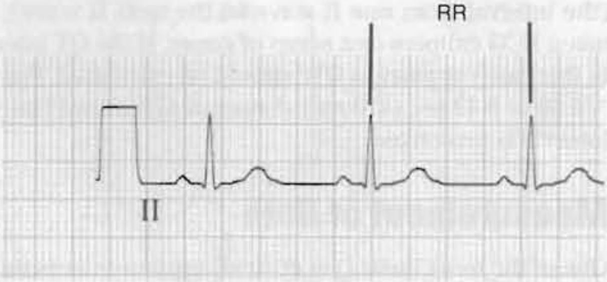


FIGURE 1.10 Heart rate 60 method. The R-R interval shown here is 0.86 sec.

Triplets (Quick, Easy, Reasonably Accurate if Rhythm Is Regular)

The triplets method is a very quick and easy calculation to perform, and as long as the rhythm is regular, it provides an estimation of HR accurate enough for most clinical purposes. First, establish that the QRS complexes are coming along at fairly regular intervals (i.e., the R-R interval is consistent). This is important because in the presence of varying R-R intervals the estimation of rate by the triplet method will vary depending on which R-R interval was chosen for measurement. The triplets can still be used in this situation, but for accurate rate estimation several representative R-R intervals would have to be measured and averaged.

The easiest way to use the triplets method is to find an R wave (or S wave) that falls on a thick (200 ms) line. The distance in big (200 ms) boxes to the next R wave (or S if an S wave was used initially) is then counted off using the "triplets":

300-150-100 75-60-50

As shown in **Figure 1.11A**, if the next R wave falls on the first thick line from the reference R wave, then the rate is 300 bpm; if it fell on the second thick line the rate would be 150 bpm, third thick line 100 bpm, and so on. Figure 1.11B shows an example where the next R wave falls on the second thick line, making the HR approximately 150 bpm.

In the example shown in **Figure 1.12**, the reference R wave falls on a thick line, and the next R wave falls on the first thin line following the fourth thick line. If the second R wave had fallen exactly on the fourth thick line the rate would have been 75, and if it had fallen on the fifth thick line the rate would have been 60. Therefore, we know that the rate is between 75 and 60 bpm. For some situations it is accurate enough to say that the rate is "between 60 and 75." If a little more precision is desired we can interpolate very simply. Since $75 - 60 = 15$ we can estimate that each small (1 mm) box represents about 3 bpm because there are five small boxes that represent the 15 beats between 75 and 60 ($15 \div 5 = 3$). Since the R wave in question is one small box short of the thick line representing 75 bpm and each box in this case is equivalent to 3 bpm, we can estimate that the HR is 72 bpm ($75 - 3 = 72$).

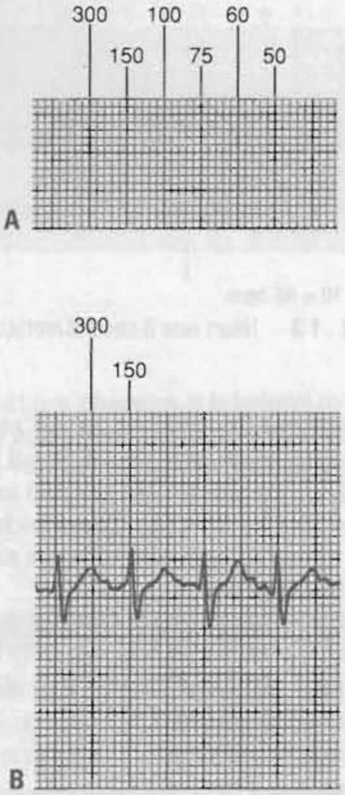

300
100
60
150
75
50
A
300
150
B

FIGURE 1.11 Heart rate triplets method. **A:** The triplets. **B:** Begin counting at the next thick line and continue until the next QRS complex.

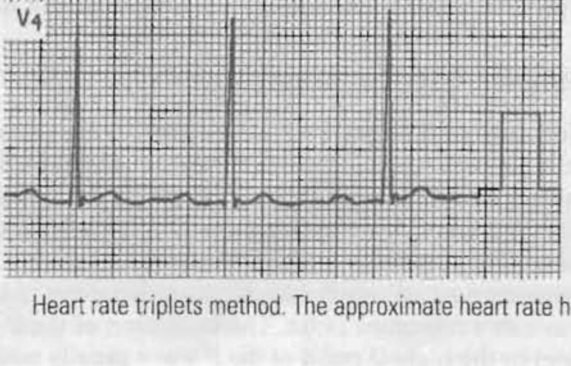
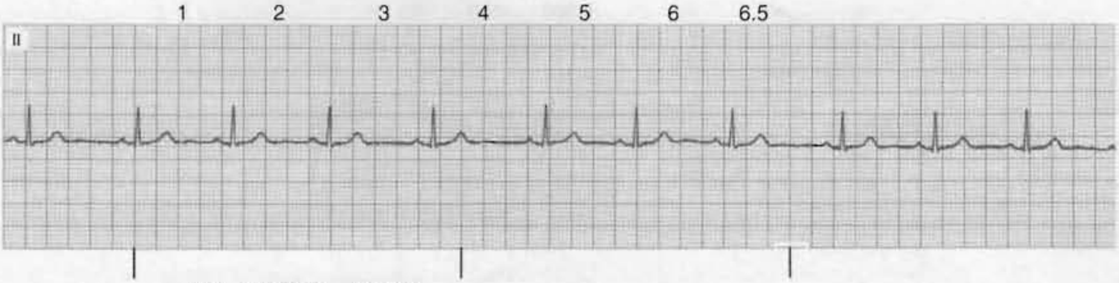

300
100
60
150
75
V4

FIGURE 1.12 Heart rate triplets method. The approximate heart rate here is 72 bpm.



HR = 6.5 × 10 = 65 bpm

FIGURE 1.13 Heart rate 6-second method.

The value assigned to each small (40 ms) box will vary. For example, if we have rates between 60 and 50 each small box represents 2 bpm (60 – 50 = 10, 10 ÷ 5 = 2), while if the rate is between 150 and 100 each small box represents 10 bpm (150 – 100 = 50, 50 ÷ 5 = 10). These techniques are fairly accurate, particularly at rates <150 bpm, but some accuracy is sacrificed for the sake of expedience, particularly at faster heart rates.

The triplet method is very good for a quick rough estimation of rate. For example, using this method it can very quickly be ascertained that the "rate is around 50" or "between 100 and 150." This is often all of the information that is needed, particularly in emergency situations.

6-Second Method (Particularly Useful if the Rhythm Is Irregular)

The 6-second method is preferred if the R-R intervals are varying, as it yields an average rate. Most ECG paper has marker lines on the bottom of the paper every 3 seconds (or sometimes every second), making it quite easy to measure a 6-second time interval. To estimate HR, one need only count (usually to the nearest half) the number of R-R intervals and then multiply by ten (add a zero). For example, in **Figure 1.13**, 6.5 R-R intervals are present in 6 seconds (note the vertical 3-second markers on the bottom of the strip); therefore, the rate is approximately 65 bpm. Note that it is R-R intervals that are counted, not R waves.

Different Atrial and Ventricular Rates

Normally a QRS complex follows each P wave, thus the atrial and ventricular rates normally are identical. Sometimes, as shown in **Figure 1.14**, atrial and ventricular rates are not the same. In this example more than one P wave appears for each QRS complex. In such cases it may be appropriate to measure both rates and describe them. The atrial rate can be measured using the same techniques described for measurement of ventricular rates. A definitive point needs to be chosen on the P wave as a reference point. The beginning of the P wave (where it leaves the baseline) or the highest point of the P wave usually makes a good reference point.

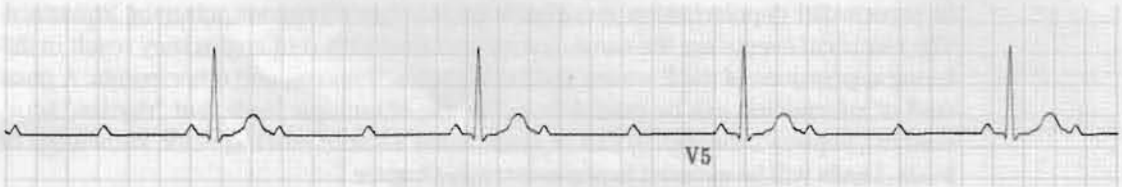


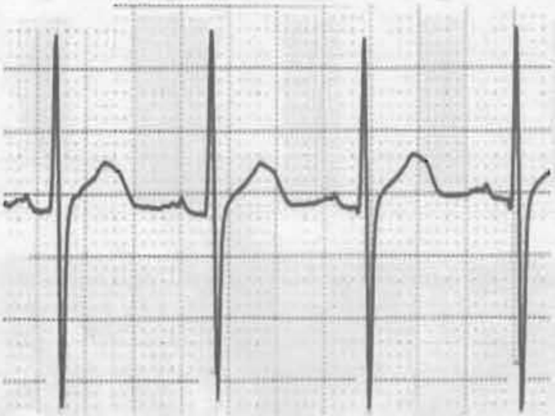
FIGURE 1.14 Differing atrial and ventricular rates. The atrial rate here is 107 bpm and the ventricular rate is 35 bpm.

Leads

Before proceeding to the next few chapters, it is helpful to have a cursory understanding of what a lead is. Simply put, a lead is an electrical view of the heart. The standard ECG consists of 12 of these views (leads), each measuring the same electrical events

CLINICAL VIGNETTE REVISITED

While working in the Stress Lab of a busy hospital, a clinical exercise physiologist receives a request for an exercise test on an inpatient. This patient is taking a medication called procainamide, which may prolong repolarization of the ventricles. The request specifies that the stress test should not be performed if the QT interval is "prolonged."



If the QT interval is more than half the R-R interval, the QT interval is probably prolonged. The QT interval of 9 mm (0.36 sec) is more than half the R-R interval of 16 mm (0.64 sec), so the physiologist calculated the QTc using the Bazett formula. The calculated QTc of 0.45 sec was greater than the 0.44-sec limit generally considered to indicate a prolonged QTc, so the attending physician was notified and the test was not performed.

of myocardial depolarization and repolarization from different points of Reference. The electrical events are the same, but viewed from different angles they result in differing appearance of the P waves, QRS complexes, T waves, and other events. A great deal of information can be gained from the use of multiple leads, but "rhythm" (covered in Chapters 2 through 6) can be understood without much specific knowledge of leads. Leads will be covered in some detail in Chapter 7.

Normal Ranges for Selected ECG Parameters

P wave duration	≤120 ms (0.12 sec)
P wave amplitude	≤0.25 mV (2.5 mm)
PR Interval	120–200 ms (0.12–0.20 sec)
QRS duration	<100 ms (0.10 sec)
QTc	<440 ms (0.440 sec)
HR	60–100 bpm

Quiz 1

1. Describe each of the QRS complexes as if speaking on the phone with a colleague.

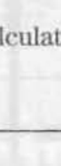
A

B

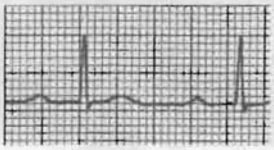
C

D

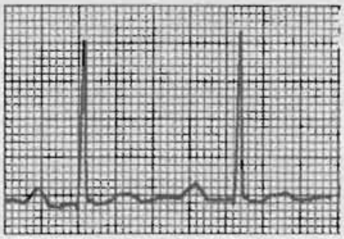
E



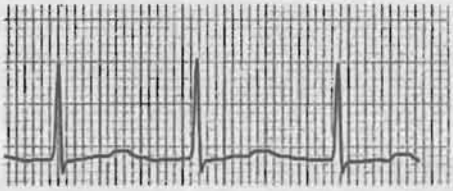
2. Measure the PR interval, QRS duration, and QT interval, and calculate a QTc. Is the QTc within normal limits?



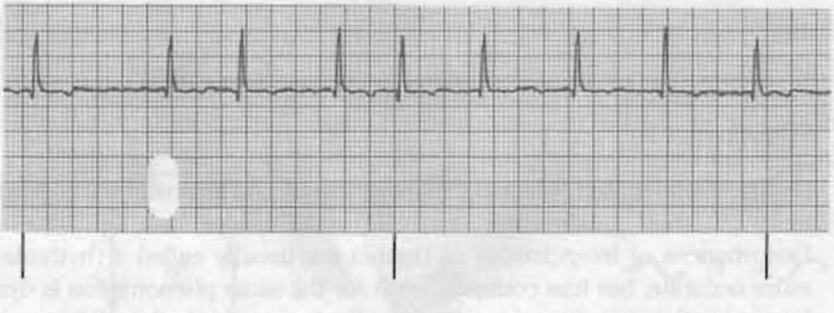
3. Using a very accurate method, calculate the HR.

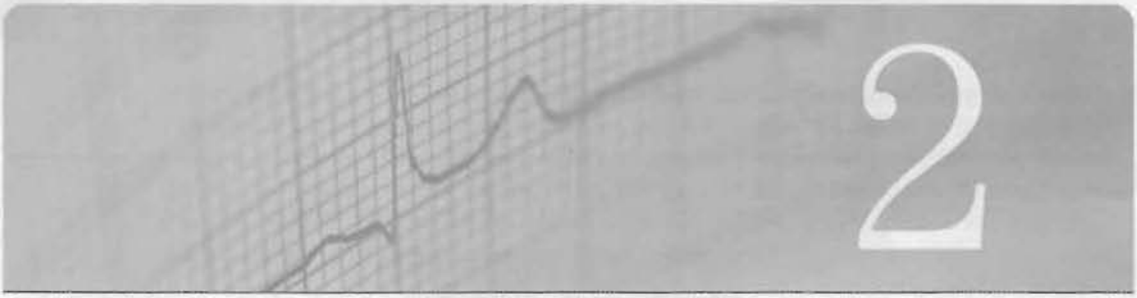


4. Measure the HR.



5. Measure the HR.





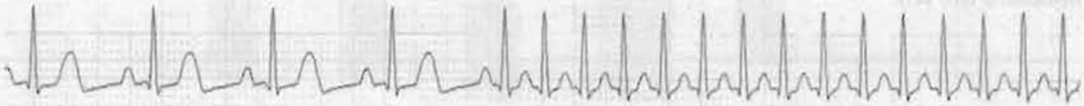
2

SUPRAVENTRICULAR RHYTHMS I

Rhythm
Sinus Rhythms
Normal Impulse Origin and Pattern of Conduction
Normal Sinus Rhythm
Sinus Arrhythmia
Sinus Tachycardia and SA Nodal Re-Entrant Tachycardia
Sinus Bradycardia
Sinus Pause
Premature Atrial Complexes
Junctional Premature Complexes
Atrial Bigeminy, Trigeminy, and Quadrigeminy
Ectopic Atrial Tachycardia
Atrioventricular Nodal Re-Entrant Tachycardia
Junctional Escape
Accelerated Junctional Rhythm

CLINICAL VIGNETTE

A new patient in phase II cardiac rehabilitation reports feeling like his "heart was pounding" while he was cooling down from his first exercise session.



Rhythm

Rhythm is the pattern of the complexes, waves, and intervals, the regularity or irregularity of their occurrence, and the relationships among these constituents. Disturbances or irregularities of rhythm are usually called arrhythmias. A perhaps more accurate, but less common, term for the same phenomenon is dysrhythmia. In keeping with usual clinical terminology, the term arrhythmia will be used in this book.

Inspection of more than one ECG lead is necessary for many diagnoses (and can sometimes be of great help in determining the rhythm), but often only one lead is needed to determine the rhythm. In the interest of simplicity and clarity, here it is assumed that the ECGs described in this section are recorded using lead II. Lead II is one of several bipolar leads, so named because these leads possess positive and negative sensing electrodes. For the current discussion the important point is that electrical current heading toward the positive pole of a lead will result in positive (rising above the baseline) deflections on the ECG, and electrical events heading away from the positive pole (and therefore toward the negative pole) will result in negative (below baseline) deflections. A current traveling perpendicular to the axis of a lead will result in a waveform with roughly equal positive and negative components. Depending on various factors, either the positive or the negative component may come first, or it may manifest as a flat line. **Figure 2.1** illustrates these concepts in relation to P waves. The same concepts apply to other waves as well.

As the positive pole of lead II is placed on the left leg, and the negative pole is located on the right arm, lead II is in line with the normal plane of key electrical events because depolarization normally tends to propagate through the heart from upper right to lower left and therefore toward the positive pole of lead II.

The discussion here begins with rhythms that have their origin above the ventricles, thus termed *supraventricular*.

Sinus Rhythms

As shown in **Figure 2.2**, the sinoatrial (SA) node is normally the first area of the heart to depolarize. (This and similar figures throughout the book are not anatomically correct representations of the heart, but rather diagrammatic illustrations of the conduction system.) As the SA node is located in the right upper portion of the right atrium, the net current spreads down and toward the left. Actually current is spreading in many directions, but the standard ECG measures the average (net) vector of the currents.

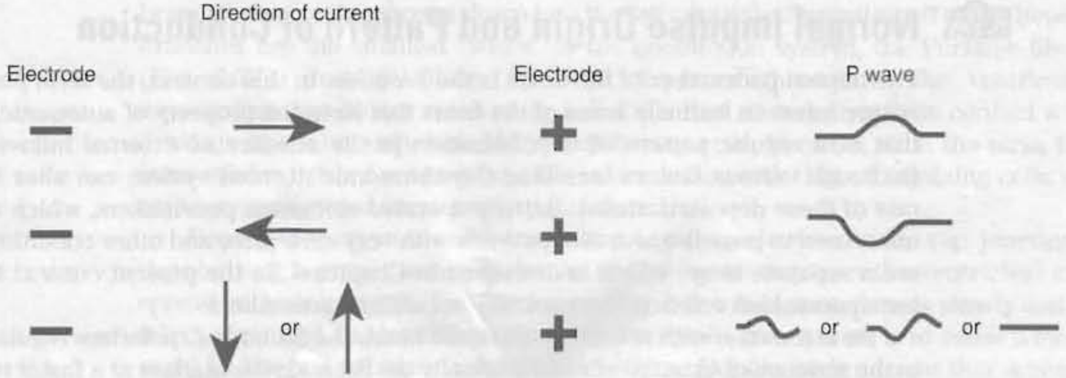


FIGURE 2.1 P waves resulting from current flow with differing orientation to electrodes.

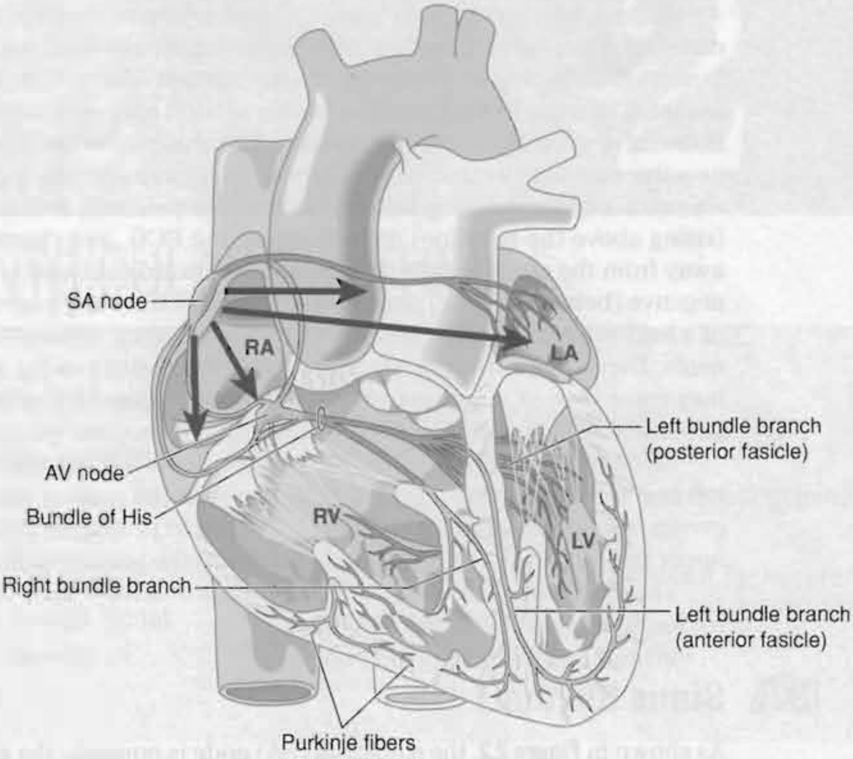

SA node
RA
LA
AV node
Bundle of His
RV
LV
Right bundle branch
Left bundle branch
(posterior fasicle)
Left bundle branch
(anterior fasicle)
Purkinje fibers

FIGURE 2.2 Atrial depolarization.

The positive pole of lead II is located on the left leg, so a P wave beginning in the SA node and spreading through the atria in the normal fashion will (in terms of the net vector of the current) be heading toward the positive pole of lead II and therefore result in a positive deflection on the cardiogram.

KSA Normal Impulse Origin and Pattern of Conduction

The normal pacemaker of the heart is the SA node. In this context, the term *pacemaker* refers to intrinsic areas of the heart that have the property of automaticity, that is, a regular pattern of depolarization in the absence of external influence (although various factors including the autonomic nervous system can alter the rate of these depolarizations). Battery-operated electronic pacemakers, which are often used to pace the hearts of patients with very slow rates and other conditions, are a separate issue, which is discussed in Chapter 5. In the present context the term pacemaker refers to the naturally occurring pacemakers.

As is the case with several areas of the heart, the SA node depolarizes regularly in the absence of external stimuli. Normally the SA node depolarizes at a faster rate than the other pacemakers so the other pacemakers remain quiescent as they are "reset" by the depolarizing current that arises from the SA node before they get a chance to fire. In the absence of depolarizations arising from the SA node, subsidiary pacemakers located in areas including the AV junction (defined below) and the ventricles can take control of pacing the heart. Usually the lower pacemakers (i.e., those located in the ventricles) have slower inherent rates; this means that the higher pacemakers (e.g., AV junction) will typically take over pacing if the SA node fails to depolarize or does so at too slow a rate. Even if the SA node is firing at a normal rate, numerous factors can excite subsidiary pacemakers, resulting in beats that do not arise from the SA node.

The normal spread of depolarization in the heart is shown in **Figure 2.3**. As previously discussed, the depolarization normally begins spontaneously in the SA node (Fig. 2.3A). The intracellular fluid of the atrial cells is in contact with that of neighboring cells via gap junctions, allowing the depolarization to spread from cell to cell throughout the atria (Fig. 2.3B). This results in the P wave of the ECG. The atrioventricular (AV) node is surrounded by atrial tissue; therefore, the wave of depolarization spreading throughout the atria reaches the AV node. Normally, the tissue separating the atria from the ventricles does not conduct current, the only normal pathway for the depolarizing current to reach the ventricles is via the AV node and the next portion of the conduction system, which is known as the bundle of His (pronounced "hiss"). The tissue of the AV node conducts current at a much slower rate than other portions of the conduction system; this results in what appears to be a slight "delay" before the current reaches the ventricles (Fig. 2.3C). Physiologically, this delay permits time for the atria to contract and further fill the ventricles with blood prior to ventricular depolarization (and subsequent contraction). The area where the AV node and the bundle of His meet is called the AV junction.

Following the "delay" at the AV node, current spreads down the bundle of His (Fig. 2.3D), and then down the left and right bundle branches (Fig. 2.3E). The bundle of His and the bundle branches are areas of tissue specialized for the conduction of current (i.e., they function as "wires" rapidly bringing the current down into the ventricles). The left bundle branch actually has two pieces, known as anterior and posterior fascicles, but for the present it is sufficient to simply think of a right bundle branch bringing the current down into the right ventricle and a left bundle branch bringing the current down into the left ventricle. Sprouting off of the bundle branches are the smallest "wires" of the conduction system, the Purkinje fibers (Fig. 2.3F). Purkinje fibers are well dispersed throughout the ventricular myocardium but do not reach every cell. Cells that are not directly in contact with Purkinje fibers are depolarized by neighboring cells, because, as in the atria, the intracellular fluid of ventricular cells is in contact with that of adjoining cells via cellular connections known as gap junctions.

Because ventricular cells are linked together electrically by gap junctions, depolarizing currents reaching the ventricles (or arising from the ventricles) can spread without using the conduction system, but would spread more slowly and in a less-organized fashion. The purpose of the conduction system is to cause a more rapid and organized ventricular depolarization. Depolarizing current that spreads via the normal conduction system usually results in a "narrow" QRS complex (i.e.,

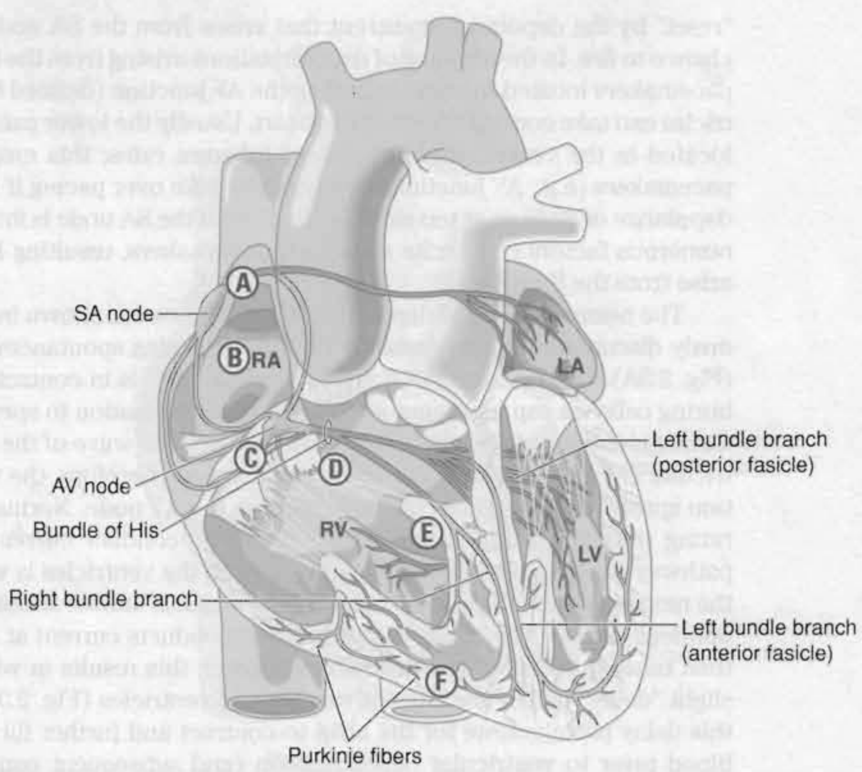

A
SA node
B RA
LA
Left bundle branch (posterior fasicle)
C
D
AV node
Bundle of His
RV
E
LV
Right bundle branch
Left bundle branch (anterior fasicle)
F
Purkinje fibers

FIGURE 2.3 Steps of the conduction system. **A:** SA node depolarizes. **B:** Depolarization spreads throughout the atria. **C:** Delay at the AV node. **D:** Bundle of His depolarizes. **E:** Left and right branches depolarize. **F:** Purkinje fibers depolarize.

<100 ms) because it travels quickly; depolarizations that do not go though the conduction system normally, but rather largely spread via the slower cell to cell pathways, result in a "wide" (i.e., ≥100 ms) QRS complex.

KSA Normal Sinus Rhythm

The normal sinus rhythm (NSR) is the "normal" rhythm of the heart. Several requirements must be met for the designation of NSR. A "normal" heart rate (HR) has been somewhat arbitrarily designated as between 60 and 100 beats per minute (bpm). As the "sinus" part of the name indicates, in this rhythm it is assumed that the SA node is pacing the heart. Therefore, a positive P wave (representing atrial depolarization) should be present in lead II. This is because, as indicated in the previous section, the net current is heading down and to the left from the SA node and, therefore, toward the positive pole of lead II. Actually many authorities believe that the P wave must be positive in leads I, II, V_5, and V_6 to truly confirm sinus rhythm.

As a practical matter, if the P wave is positive in lead II, it usually (but not always) is positive in leads I, V_5, and V_6.

In addition to the above requirements, the rhythm must be regular and one P wave must be present for each QRS complex. **Figure 2.4** shows examples where all of these conditions are met. The two short strips of Figure 2.4A show simultaneous recordings in lead II, and a lead called aVR. Notice that the P waves are positive in lead II and negative in lead aVR. The positive pole of lead aVR is located on the right arm; therefore, current moving down and to the left should be seen as a negative deflection in lead aVR as it is traveling away from the positive pole of this lead. If normal sinus rhythm is present the P waves should be positive in lead II and negative in lead aVR. In the absence of technical error, if the P waves are positive in lead II it is virtually ensured that they will be negative in aVR. The longer lead II strip in Figure 2.4B illustrates the regularity of the rhythm and the consistent presence of one P wave for each QRS complex. Longer strips such as this one are commonly referred to as rhythm strips.

Sinus Arrhythmia

If the other conditions for NSR are present (HR between 60 and 100 bpm, positive P waves in lead II, and one P wave for each QRS complex), but the rhythm is irregular (i.e., the R-R interval is varying), then sinus arrhythmia is present. Compare Figures 2.4 and **2.5**. Notice with NSR (Fig. 2.4), the R-R interval shows little variation. In other words, the rhythm is regular. Contrast this with Figure 2.5, where the R-R interval varies significantly. One commonly used definition of significant

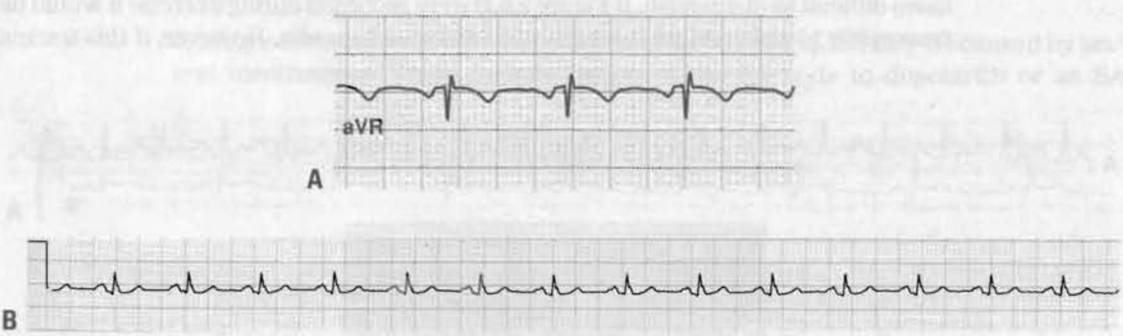


FIGURE 2.4 Normal sinus rhythm. **A:** P waves are positive in lead II and negative in lead aVR. **B:** Rhythm is regular with a rate between 60 and 100 bpm. PR intervals are consistent with one P wave for each QRS.

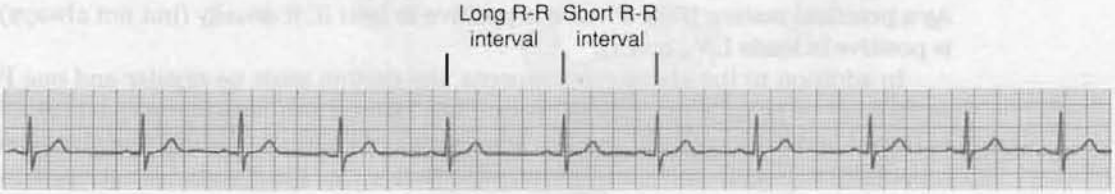


FIGURE 2.5 Sinus arrhythmia.

variation is a difference of at least 80 ms (two small boxes on the ECG paper) between the shortest and longest R-R intervals.

Some variation in the R-R interval is normal and often is associated with respiration. With inspiration the diaphragm descends, decreasing intrathoracic pressure, leading to less vagal stimulation. This in turn increases HR. The opposite effect occurs with exhalation; vagal stimulation is increased and HR is decreased. It is quite normal for HR to increase (decreased R-R interval) with inspiration and decrease (lengthened R-R interval) with expiration. Sinus arrhythmia is simply an exaggeration of this normal variation. Sinus arrhythmia that is associated with ventilation is sometimes termed *phasic sinus arrhythmia*. Sometimes sinus arrhythmia is not related to ventilation; this can be referred to as a *nonphasic sinus arrhythmia*.

KSA Sinus Tachycardia and SA Nodal Re-Entrant Tachycardia

If all of the conditions for NSR are present, with the exception that the HR is >100 bpm, then sinus tachycardia is present **(Fig. 2.6)**. This term simply indicates a fast heart rate. As the heart rate increases it may become more difficult to discern P waves. In Figure 2.6A, P waves are clearly visible. In Figure 2.6B, the rate is faster and the P waves have begun to merge with the T waves of the preceding beats, making them more difficult to distinguish. If Figure 2.6.B were recorded during exercise it would be reasonable to assume that it represented sinus tachycardia. However, if this tracing

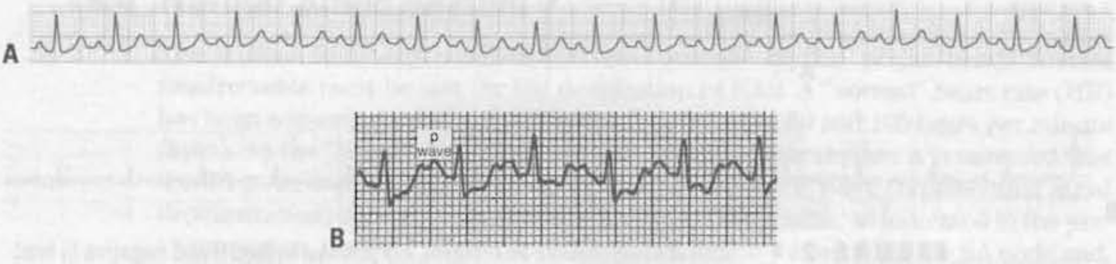


FIGURE 2.6 Sinus tachycardia. **A:** The heart rate is approximately 123 bpm. **B:** The heart rate is approximately 150 bpm.

were obtained under resting conditions, it likely would represent another sinus rhythm, SA nodal re-entrant tachycardia, an abnormal depolarizing current that spins rapidly around in the area of the SA node resulting in fast heart rates. As the current circles around repeatedly re-entering the SA node it is called a re-entrant, or circuit, current. Since the initial depolarizations come from the SA node, the P waves appear normal. The rate is typically >100 bpm, and the rhythm is regular.

Sinus tachycardia usually comes on gradually, for example, the rate may increase from 70 to 80 to 90 and so on until it exceeds 100 bpm. Sinus tachycardia also shows a gradual decline in rate when returning to NSR. In contrast, SA nodal re-entrant tachycardia has a sudden onset and a sudden cessation. For example, the rate may jump in one beat from 70 to 150 bpm. The termination is equally abrupt. If the onset or termination of the tachycardia is witnessed the diagnosis is simplified. If the rhythm shown in Figure 2.6 had a sudden onset and sudden termination this would virtually ensure that it is an SA nodal re-entrant tachycardia. Gradual onset and termination of the same rhythm implies sinus tachycardia. If the onset is not witnessed, the rate and regularity offer important clues. Higher rates and constant R-R intervals more likely represent SA nodal re-entrant tachycardia, lower rates and R-R variability favor a diagnosis of sinus tachycardia. In cases wherein the rhythm cannot be discerned with certainty, the term *versus* is often inserted between the possibilities, so Figure 2.6B might be described as "sinus tachycardia versus SA nodal re-entrant tachycardia."

KSA Sinus Bradycardia

Sinus bradycardia **(Fig. 2.7)** is simply a slow HR, usually defined as <60 bpm. Other than the low rate, the criteria for NSR are met. The term *marked sinus bradycardia* is sometimes used when the rate is very low, as in Figure 2.7B. Slow rates can indicate pathology, but are also seen in highly trained endurance athletes (due to high vagal tone) and patients taking beta adrenergic antagonist medications (commonly known as beta blockers).

KSA Sinus Pause

A delay occurring before the appearance of a P wave **(Fig. 2.8)** may be caused by several mechanisms. These include failure of the SA node to depolarize or an SA

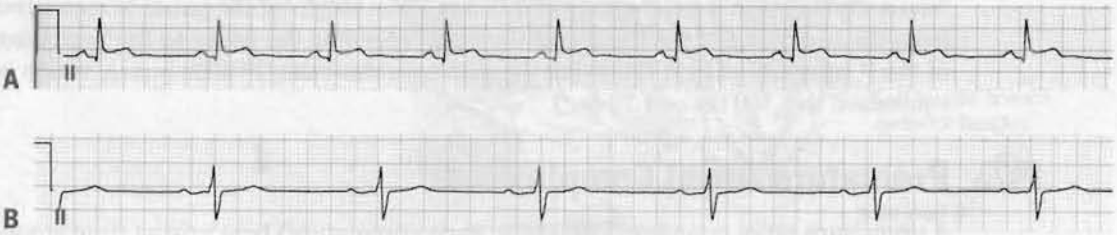
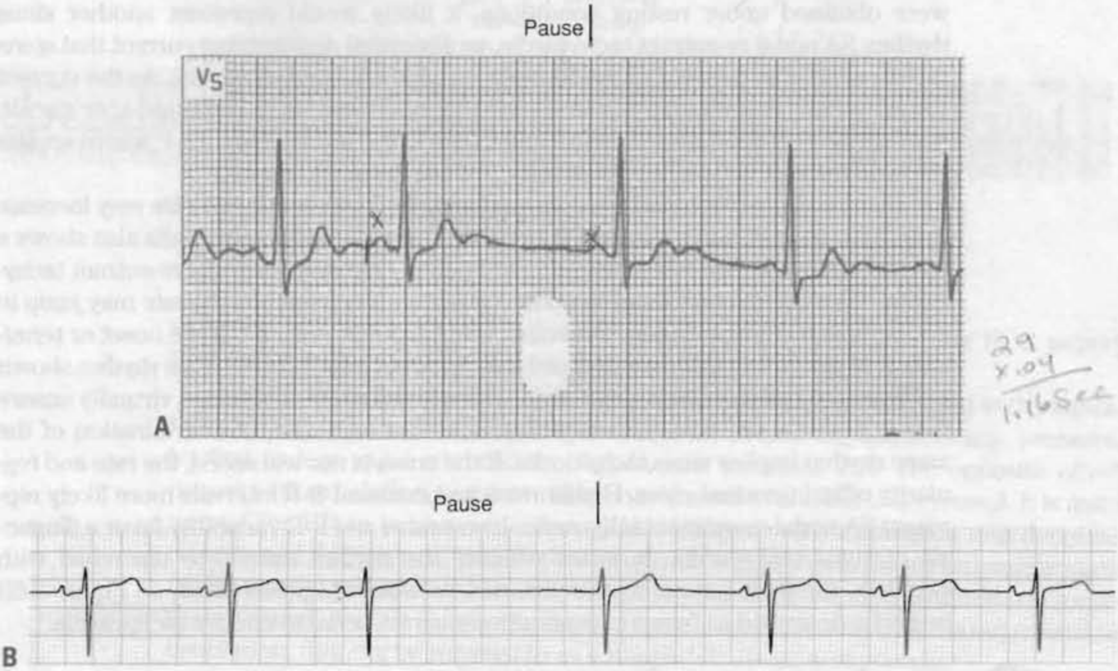


FIGURE 2.7 Sinus bradycardia. **A:** The heart rate is approximately 55 bpm. **B:** The heart rate is approximately 42 bpm.



29
x.04
1.16sec

FIGURE 2.8 Sinus pause. **A:** The rhythm resumes with the sinus P wave. **B:** The rhythm resumes with the ectopic beat.

"block," which does not permit the depolarization to escape from the SA node. It is often difficult, if not impossible, to determine the mechanism of the delay from a standard ECG. Regardless of mechanism, the general term sinus pause can be used to describe this phenomenon.

Note in Figure 2.8A that after the pause the rhythm resumes with a P wave that looks like the normal P waves. In Figure 2.8B the rhythm resumes with a normal appearing QRS that is not preceded by a P wave. As discussed in the following sections, this implies that the beat does not originate in the SA node.

Pauses can be quantified. For example, Figure 2.8A could be described as "sinus rhythm with a sinus pause of 1.16 sec." The length of the pause is measured from the beginning of the P wave of the beat preceding the pause to the beginning of the P wave of the beat following the pause (in this case, 29 little boxes, which is equivalent to 1,160 ms or 1.16 sec).

KSA Premature Atrial Complex

A premature atrial complex (PAC) is an early (premature) beat arising from somewhere above the ventricles other than the sinus node (i.e., in the atria or the AV junction). In general, depolarizations arising in areas of the heart other than the normal pacemaker (SA node) are referred to as *ectopic.* The site of origin of these ectopic depolarizations is termed an ectopic focus.

Early beats can arise from numerous areas of the heart. A premature depolarization arising from above the ventricles (supraventricular) can be called by many names, perhaps the most common is PAC. Other terms used to describe these beats include atrial premature contraction (APC), atrial premature depolarization (APD), and atrial premature beat (APB). The term junctional premature complex (JPC) is also sometimes used to describe certain supraventricular beats.

A PAC starts in an ectopic focus somewhere in the atria or the AV junction. Since the P wave represents atrial depolarization, it follows that depolarizations arising in areas of the atria other than the SA node will typically have P waves that differ in appearance from the sinus P waves. They will usually also take a different amount of time to travel through the atria and down into the ventricles, thus the PR intervals of PACs may differ from that of sinus beats. A PAC originating in the upper or middle parts of the atria may still result in an upright P wave in lead II. This is because the mean electrical vector of atrial depolarization is generally heading toward the positive pole of lead II (**Fig. 2.9**). In order to be a PAC the beat must

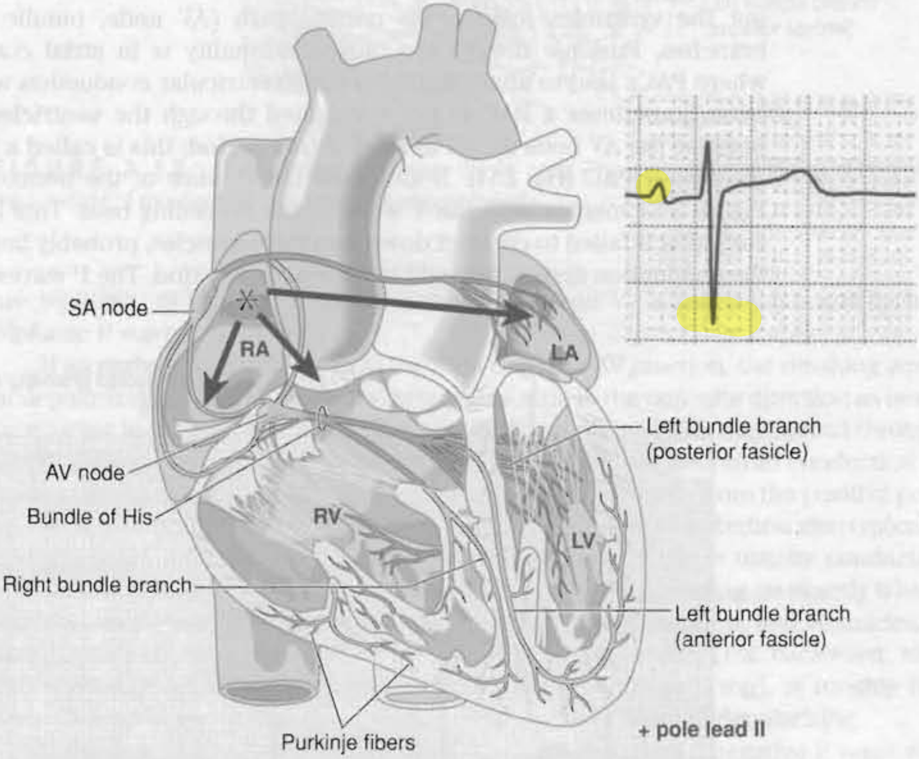


FIGURE 2.9 Atrial ectopic focus. The corresponding ECG is shown at the top right of this figure (* indicates the ectopic focus).

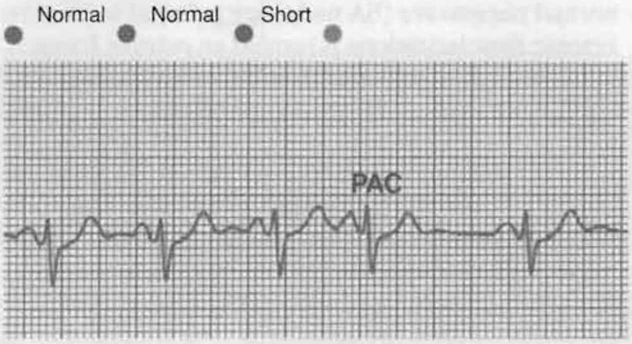


FIGURE 2.10 Premature atrial complex (PAC). P-P intervals are shown at the top left of the strip.

be early; notice how the P-P interval of the PAC shown in **Figure 2.10** is shorter than the other P-P intervals. This is just a way of indicating that this P wave came early.

Typically a PAC is conducted normally through the AV node and ventricles, resulting in a normal appearing QRS complex. This is because conduction throughout the ventricles follows the normal path (AV node, bundle of His, bundle branches, Purkinje fibers), the only abnormality is in atrial conduction. Cases where PACs lead to abnormal (aberrant) ventricular conduction will be discussed later. Sometimes a PAC is not conducted through the ventricles at all, usually because the AV node is still in a refractory period; this is called a blocked or nonconducted PAC (**Fig. 2.11**). Notice how the P wave of the nonconducted PAC in Figure 2.11 merges with the T wave of the preceding beat. This P wave came so early that it failed to conduct down into the ventricles, probably because the rest of the conduction system was still in a refractory period. The P waves in this example

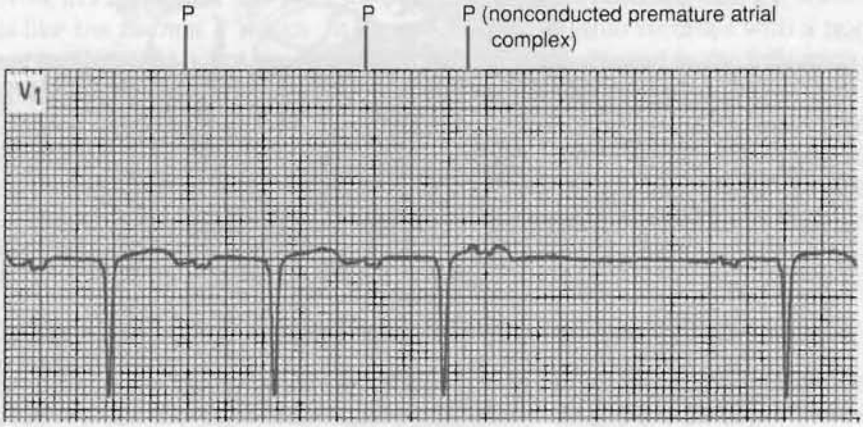


FIGURE 2.11 Nonconducted (blocked) premature atrial complex.

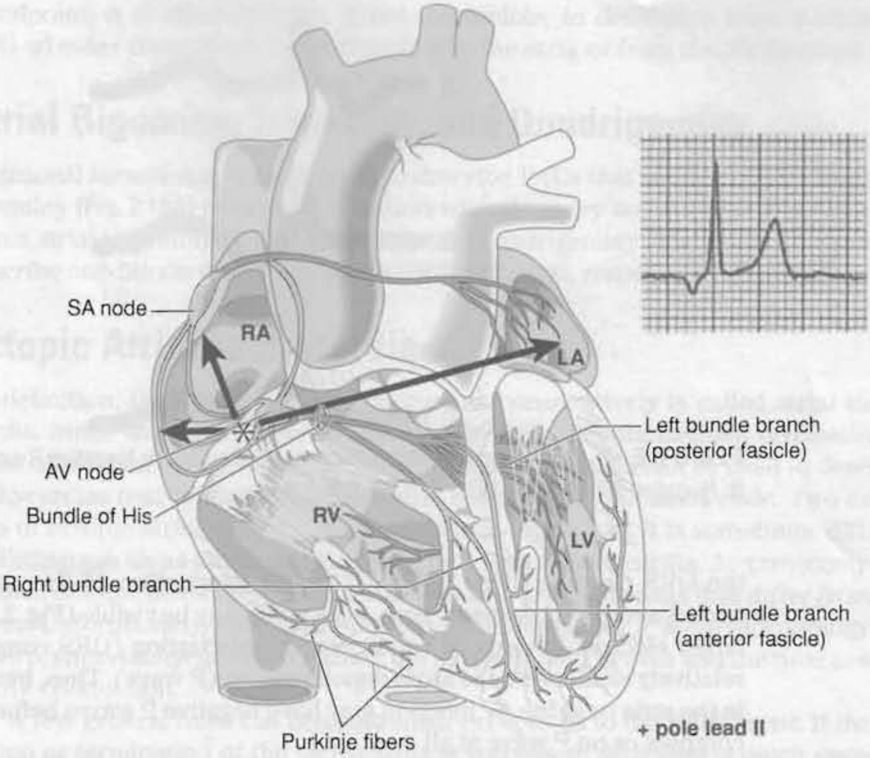


FIGURE 2.12 Atrial ectopic focus near the AV node. The corresponding ECG is shown at the top right of this figure (* indicates the ectopic focus).

are biphasic, as this strip was recorded using lead V_1, a lead that normally has biphasic P waves.

If an early beat arises lower in the atria or at the AV junction, the resulting wave of depolarization typically spreads through the atria in the opposite direction as beats originating in the SA node **(Fig. 2.12)**. In other words, these PACs may spread through the atria from bottom to top, as opposed to the normal top to bottom conduction of sinus beats. In this case atrial depolarization is spreading away from the positive pole of lead II, resulting in a negative P wave in that lead. The depolarization also typically spreads down into the ventricles; however, here the impulse is usually conducted normally, resulting in a normal appearing QRS complex. Depending on exactly where the PAC arises and the prevailing conditions in the atria, junction, and ventricles, it may happen that the atria depolarize ahead of the ventricles (but backward, also called retrograde, in that the impulse spreads from bottom to top), at roughly the same time as the ventricles, or after the ventricles have finished depolarizing.

If the depolarization spreads through the atria first, then a negative P wave will be seen before the QRS complex **(Fig. 2.13A)**; if the depolarization spreads through the ventricles first and then through the atria, a negative P wave will be seen after

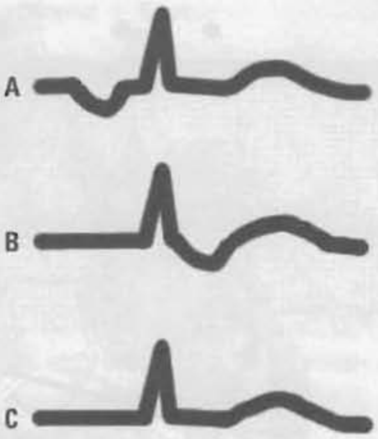


FIGURE 2.13 P waves with retrograde conduction. **A:** Negative P wave before QRS. **B:** Negative P wave after QRS. **C:** No visible P wave.

the QRS complex (Fig. 2.13B). If the depolarization of the atria and ventricles occurs at roughly the same time, no P wave may be visible (Fig. 2.13C), as the much larger electrical events of ventricular depolarization (QRS complex) obscure the relatively small events of atrial depolarization (P wave). Thus, beats originating low in the atria or at the AV junction may have negative P waves before or after the QRS complex or no P wave at all.

Junctional Premature Complexes

Figure 2.14 shows a PAC with a negative P wave prior to the QRS complex. Premature beats with negative or missing P waves are sometimes referred to as JPCs because many of them arise at or near the AV junction. From a practical

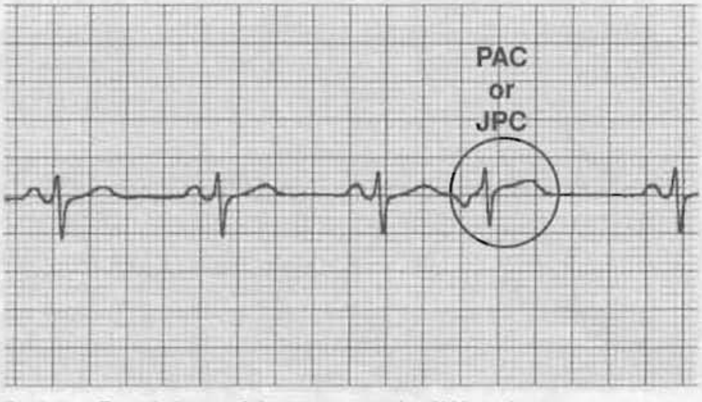


FIGURE 2.14 Ectopic beat arising at or near the AV junction.

standpoint, it is often difficult, if not impossible, to determine from a standard ECG whether these beats arose from low in the atria or from the AV junction.

Atrial Bigeminy, Trigeminy, and Quadrigeminy

Additional terminology can be used to describe PACs that occur in patterns. Atrial bigeminy **(Fig. 2.15A)** refers to a situation wherein every second beat is a PAC. The terms atrial trigeminy (Fig. 2.15B) and atrial quadrigeminy (Fig. 2.15C) are used to describe conditions where every third or fourth beat, respectively, is a PAC.

Ectopic Atrial Tachycardia

By definition, three or more PACs occurring consecutively is called atrial tachycardia. Since the term *ectopic* is used to describe depolarizations originating in areas other than the sinus node, ectopic atrial tachycardia can be used to describe tachycardias that originate from the atria but not from the sinus node. Two examples of ectopic atrial tachycardia are shown in **Figure 2.16**. It is sometimes difficult to distinguish sinus tachycardia from ectopic atrial tachycardia. As previously discussed, ectopic beats usually have P waves and/or PR intervals that differ from the norm. Unfortunately, even if the normal PR interval and P wave morphology are known, sinus tachycardia may affect the shape of the P waves and the time needed for AV conduction.

A few general rules can help, although exceptions to the rules occur. If the initiation or termination of the tachycardia is witnessed, diagnosis is much easier as ectopic atrial tachycardias typically have a sudden onset and termination. Notice in Figure 2.16B how the rate abruptly changes. The initial rhythm is sinus tachycardia. A PAC then initiates a short run of ectopic atrial tachycardia. Notice how the P wave of the first ectopic beat merges with the T wave of the proceeding beat. The

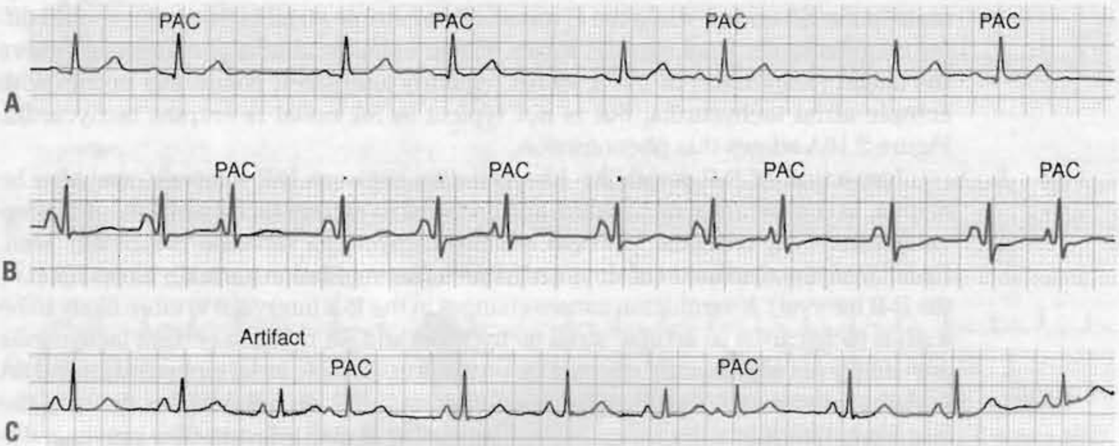


FIGURE 2.15 PAC patterns. **A:** Atrial bigeminy. **B:** Atrial trigeminy. **C:** Atrial quadrigeminy.

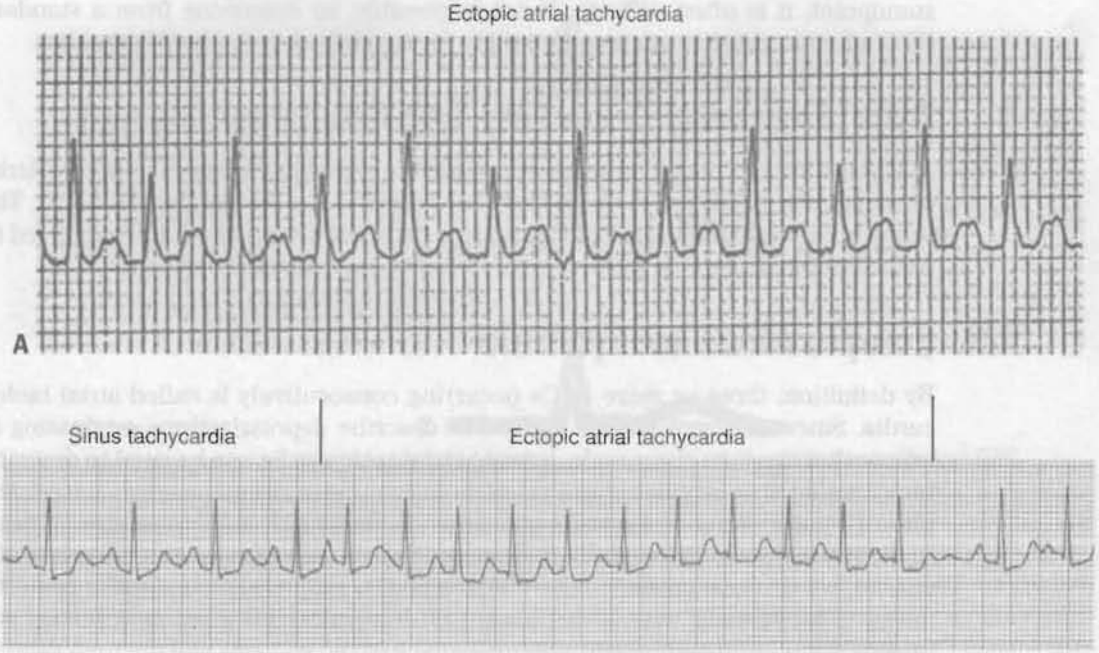


FIGURE 2.16 Ectopic atrial tachycardia. **A:** Note the alteration in the height of the R waves (electrical alternans). **B:** The sudden change in rate at the onset and termination helps to distinguish this ectopic atrial tachycardia from sinus tachycardia.

P waves are then not readily apparent (but are present on top of the T waves) until the sinus P waves re-emerge toward the end of the strip. Sinus tachycardia typically has a more gradual onset and termination. An exception to this is the previously described SA nodal re-entrant tachycardia, which has a sudden onset. The P waves of an SA nodal re-entrant tachycardia usually resemble sinus P waves (as they originate in the SA node), while the P waves of an ectopic atrial tachycardia should differ in appearance from sinus P waves. Electrical alternans, a phenomenon where the height (amplitude) of the R waves regularly alternates, commonly occurs with ectopic atrial tachycardia, but is not typical in SA nodal re-entrant tachycardia. Figure 2.16A shows this phenomenon.

Inspection of R-R variability (the variation between R-R intervals) can also be helpful. In ectopic atrial tachycardia and SA nodal re-entrant tachycardia the R-R interval is often very constant, whereas in sinus tachycardia variation is typically seen. Recall from the discussion of sinus arrhythmia that ventilation normally alters HR (i.e., the R-R interval). If ventilation causes changes in the R-R interval it is more likely to be a sinus tachycardia as ectopic atrial tachycardia and SA nodal re-entrant tachycardia are usually not significantly affected by ventilation. Ectopic atrial tachycardias and SA nodal re-entrant tachycardias often exhibit rates >160 bpm; the higher the rate, the less likely that it is sinus tachycardia. The setting is also relevant. The previous discussion assumes that the patient is at rest. During strenuous exercise sinus rates as high as 180 to 200 bpm are commonly observed in younger people, but it is unusual (although possible) for sinus tachycardia to have a rate of >140 to 160 bpm at rest.

Atrioventricular Nodal Re-Entrant Tachycardia

Current can rapidly circulate around the AV node, similar to what happens near the SA node in SA nodal re-entrant tachycardia. As might be expected, if the re-entry is occurring around the AV node, the resulting tachycardia is called AV nodal re-entrant tachycardia (AVNRT). The P waves are usually not seen or are negative and follow the QRS complex.

Figure 2.17 A shows an ectopic atrial tachycardia and two variations of AVNRT (Fig. 2.17B,C). Notice that the ectopic atrial tachycardia has negative P waves visible before the QRS complexes (recall that ectopic atrial tachycardias can have positive or negative P waves). Contrast this with the two AVNRTs where P waves either are not visible (Fig. 2.17C) or are negative and follow the QRS complexes (Fig. 2.17B).

It is common practice to refer to any of these three rhythms as junctional tachycardia. Because the mechanisms underlying the rhythms are different, it is preferable to distinguish between ectopic atrial and atrioventricular re-entrant tachycardia.

Junctional Escape

As previously discussed, areas near the AV junction possess the ability to spontaneously depolarize. If left alone the "junction" will rhythmically depolarize at a rate of around 40 to 60 times per minute. Usually the SA node depolarizes at a faster rate. As each sinus depolarization resets the other pacemakers of the

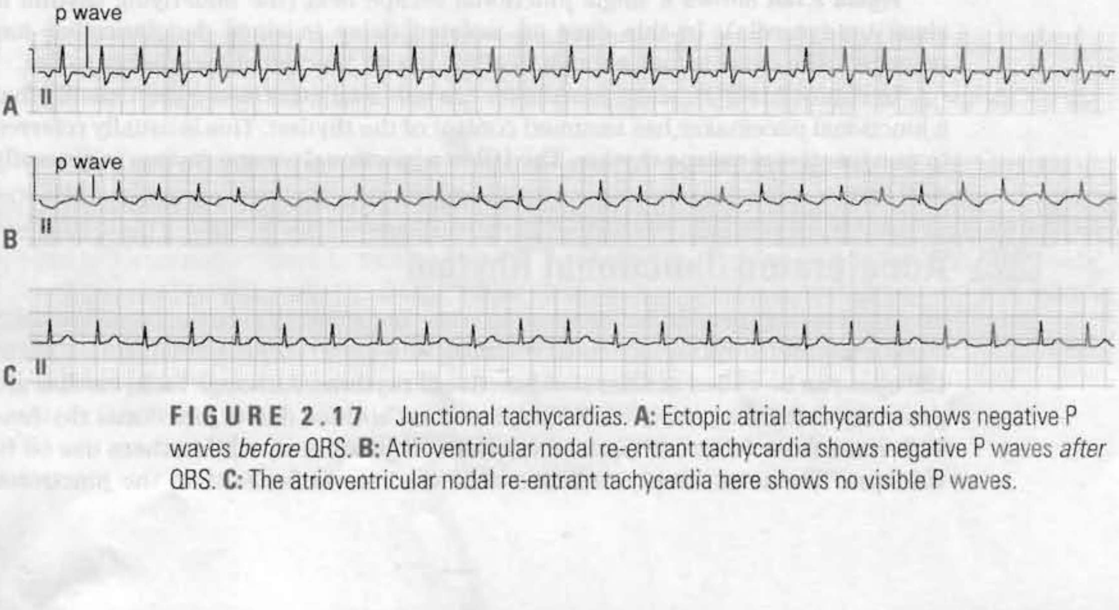


FIGURE 2.17 Junctional tachycardias. **A:** Ectopic atrial tachycardia shows negative P waves *before* QRS. **B:** Atrioventricular nodal re-entrant tachycardia shows negative P waves *after* QRS. **C:** The atrioventricular nodal re-entrant tachycardia here shows no visible P waves.

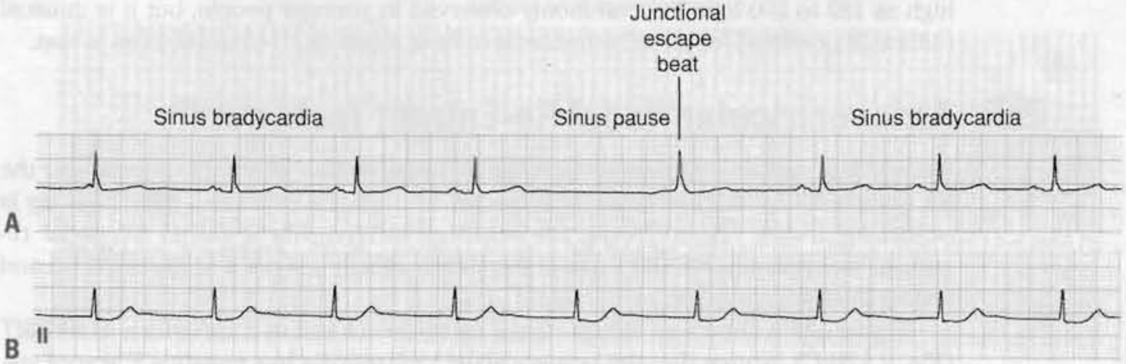


FIGURE 2.18 **A:** Junctional escape beat. **B:** Junctional escape rhythm.

heart, secondary pacemakers such as the AV junction will not typically become apparent unless the SA node fails to depolarize or does so at an abnormally slow rate.

The morphology of junctional escape beats is identical to what was described earlier for early beats arising from at or near the AV junction. The difference is that these beats are not early. In fact they could be thought of as late, as they usually only occur if the SA node or other higher pacemaker fails to fire. This is a protective mechanism; in the event that the SA node fails to depolarize the heart in a timely fashion these and other subsidiary pacemakers can take over.

As previously discussed, depolarizations arising at or near the AV junction will typically have a normal QRS complex and either (a) no apparent P wave, (b) a negative P wave before the QRS complex, or (c) a negative P wave after the QRS complex. The QRS complexes are typically normal, as ventricular depolarization occurs via the normal ventricular conduction system.

Figure 2.18A shows a single junctional escape beat (the underlying rhythm is sinus bradycardia). In this case an isolated delay in sinus depolarization has occurred. The delay is long enough to allow the AV junctional pacemaker to fire.

In Figure 2.18B the sinus pacemaker has either failed or slowed sufficiently that a junctional pacemaker has assumed control of the rhythm. This is usually referred to as a junctional escape rhythm. The HR in a junctional escape rhythm is generally <60 bpm.

KSA Accelerated Junctional Rhythm

Rhythms that appear to be originating at or near the AV junction (narrow QRS complexes and negative or absent P waves) and have rates between 60 and 100 to 120 bpm can be called accelerated junctional rhythms. Although tachycardias are generally defined as rates >100 bpm, some authorities define junctional rhythms with rates from 60 to 120 bpm as accelerated junctional, while others use 60 to 100 bpm. The term simply indicates that the rate is faster than the junctional

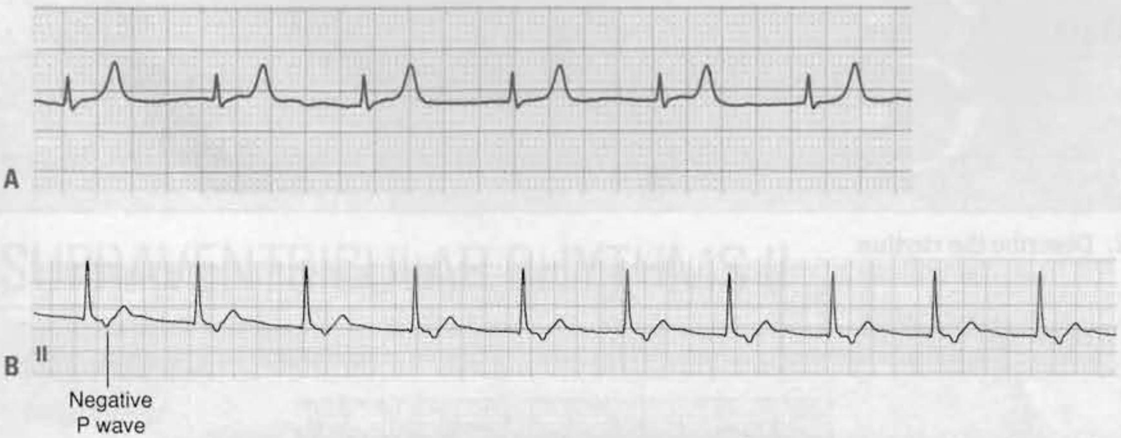
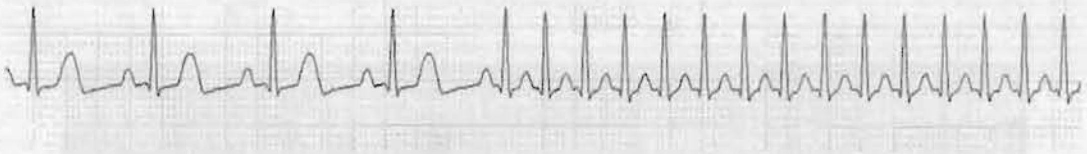


FIGURE 2.19 Accelerated junctional rhythms. **A:** Shows an accelerated junctional rhythm with a rate of about 70 bpm and no apparent P waves. **B:** Shows an accelerated junctional rhythm with a rate of about 64 bpm and negative P waves after QRS.

escape rate, yet slower than a junctional (ectopic atrial) tachycardia. Two examples are shown in **Figure 2.19**. As with other junctional rhythms, P waves may be absent or negative.

CLINICAL VIGNETTE REVISITED

A new patient in phase II cardiac rehabilitation reports feeling like his heart was pounding while he was cooling down from his first exercise session.

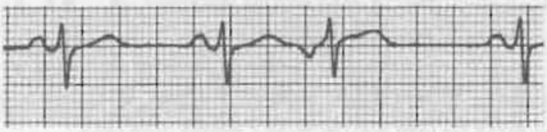


A brief run of supraventricular tachycardia, which most likely was atrioventricular re-entrant tachycardia, was noted on the telemetry monitor by the physiologist. The patient was stable and in no distress after the episode. Blood pressure was taken and the patient queried about any associated symptoms, changes in medications, habits, activities, or other factors, then the patient's physician was contacted for instructions.

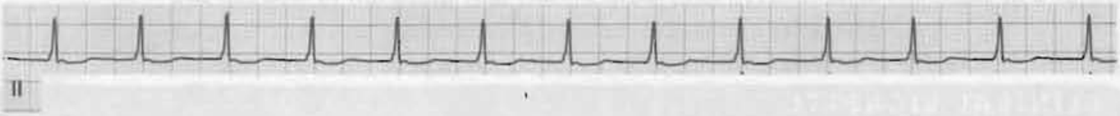
Quiz 2

1. Describe the normal sequence of myocardial depolarization.

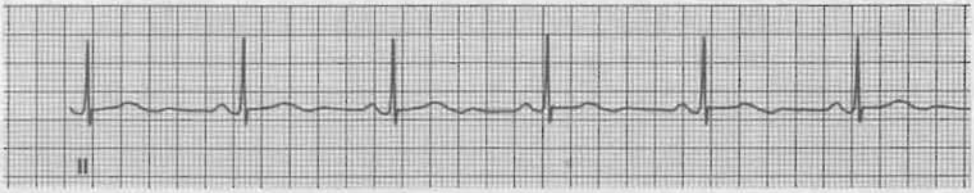
2. Describe the rhythm.

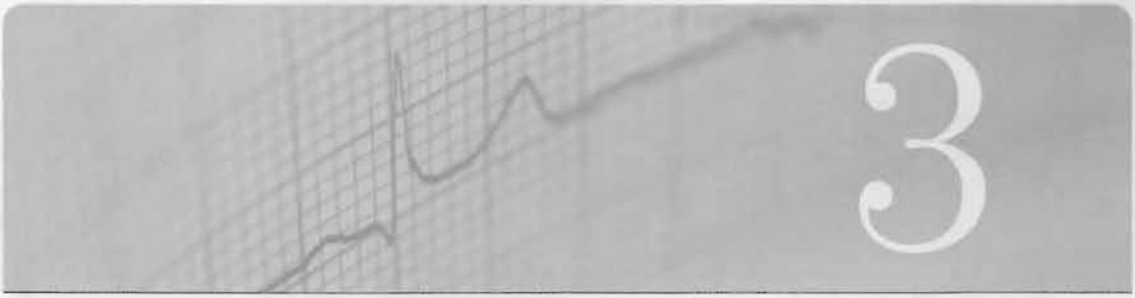


3. Describe the rhythm.



4. Describe the rhythm.





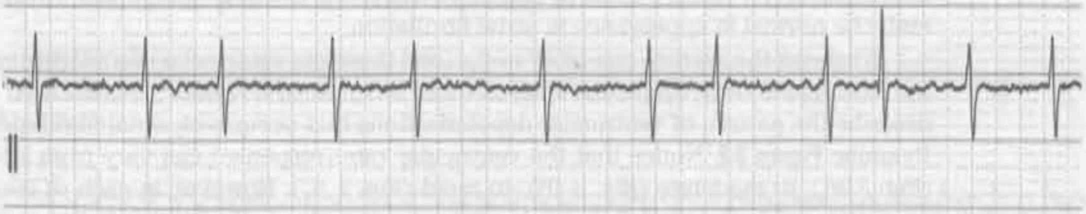
3

SUPRAVENTRICULAR RHYTHMS II

Atrial Fibrillation
Atrial Flutter
Wandering Atrial Pacemaker and Multifocal Atrial Tachycardia

CLINICAL VIGNETTE

A patient in phase III cardiac rehabilitation who previously has had a regular pulse describes feeling a "lot of skipped beats" while taking her pulse during cool down after exercise. A rhythm strip is taken and shows what appears to be a lot of artifact and a very irregular rhythm.



Atrial Fibrillation

In atrial fibrillation (A-Fib), electrical activity is so chaotic that no organized contractile activity is occurring in the atria. This results in atria that are quivering rather than contracting and the normal contribution of atrial contraction to ventricular filling is lost. A normal P wave comes from the organized spread of electrical activity throughout the atria, thus the P wave is also lost. During atrial fibrillation, hundreds of areas of the atria are depolarizing independently and in various directions so the electrocardiogram shows no signs of regularly occurring organized atrial electrical activity. Instead of P waves fibrillatory, or f waves, may be seen. These f waves **(Fig. 3.1)** may vary from very fine undulations (Fig. 3.1A)

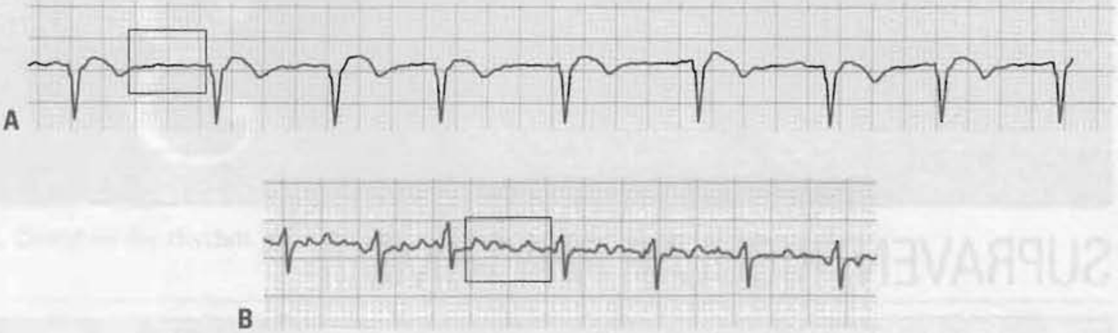


FIGURE 3.1 F waves. **A:** Fine f waves. **B:** Coarse f waves.

where the baseline appears almost flat, to coarse waves that may occasionally look almost like P waves (Fig. 3.1B).

A major determinate of clinical status in the patient with atrial fibrillation is the ventricular rate, which is determined by how many of the hundreds of atrial impulses are able to depolarize the atrioventricular (AV) node. Since the impulses are coming at the AV node from a variety of directions, and at random times, conduction down into the ventricles is quite haphazard. However, when the AV node is depolarized, conduction from that point will usually occur through the normal mechanisms (AV node, bundle of His, etc.), therefore, the QRS complexes will generally be normal in appearance in atrial fibrillation.

Whatever the average rate of AV nodal (and therefore ventricular) depolarization, the rhythm will be irregular. An unusual term, *irregularly irregular*, is often used to describe the pattern of ventricular depolarizations that occur with atrial fibrillation. Examine **Figure 3.2**. Notice that the ventricular rate (response) can vary from slow (Fig. 3.2A), to moderate (Fig. 3.2B), to rapid (Fig. 3.2C). However, in each of these cases the ventricular rhythm is very irregular. In many dysrhythmias the rhythm is not regular, but is irregular in a fairly predictable manner. For example, in atrial trigeminy every third beat is early. Thus, the pattern in atrial trigeminy could be called "regularly" irregular in that the irregularity is occurring in a pattern. Contrast this with the examples in Figure 3.2. In atrial fibrillation the pattern is "irregularly" irregular, as no predictable pattern of ventricular activity is present. This sometimes is not initially obvious at faster rates. On first glance the pattern of QRS complexes in Figure 3.2C may appear fairly regular. Careful measuring of the R-R intervals will reveal that this is not the case; the pattern is in fact quite irregular. The faster rate simply makes it somewhat more difficult to immediately discern this. Checking the regularity of the R-R intervals is very important in distinguishing atrial fibrillation from other rhythms.

Atrial Flutter

During atrial flutter the atria are depolarized in an organized cyclical manner and at a very high rate resulting in a distinctive "sawtooth" pattern. The F waves of atrial

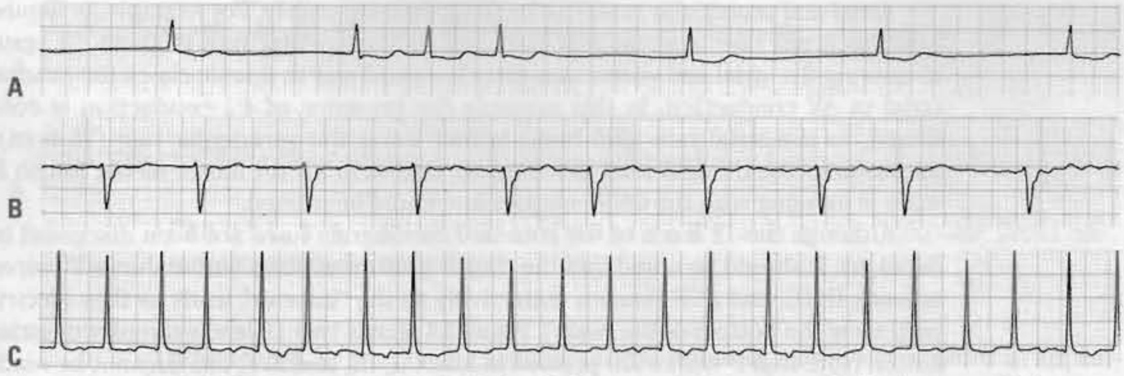


FIGURE 3.2 Atrial fibrillation. **A:** Slow ventricular response. **B:** Moderate ventricular response. **C:** Rapid ventricular response.

flutter are shown in **Figure 3.3.** Atrial fibrillation has fibrillatory waves, abbreviated with a lower case f, while an upper case F is used to abbreviate the sawtooth flutter waves of atrial flutter.

During atrial flutter the atrial rate is often close to 300 beats per minute (bpm). The ventricular rate may vary. In some cases a consistent fraction of the F waves conduct down into the ventricles; Figure 3.3A–C show consistent patterns of every fourth (4:1) or second (2:1) atrial impulse being conducted. Sometimes the AV conduction varies, as is seen in Figure 3.3D and 3.3E.

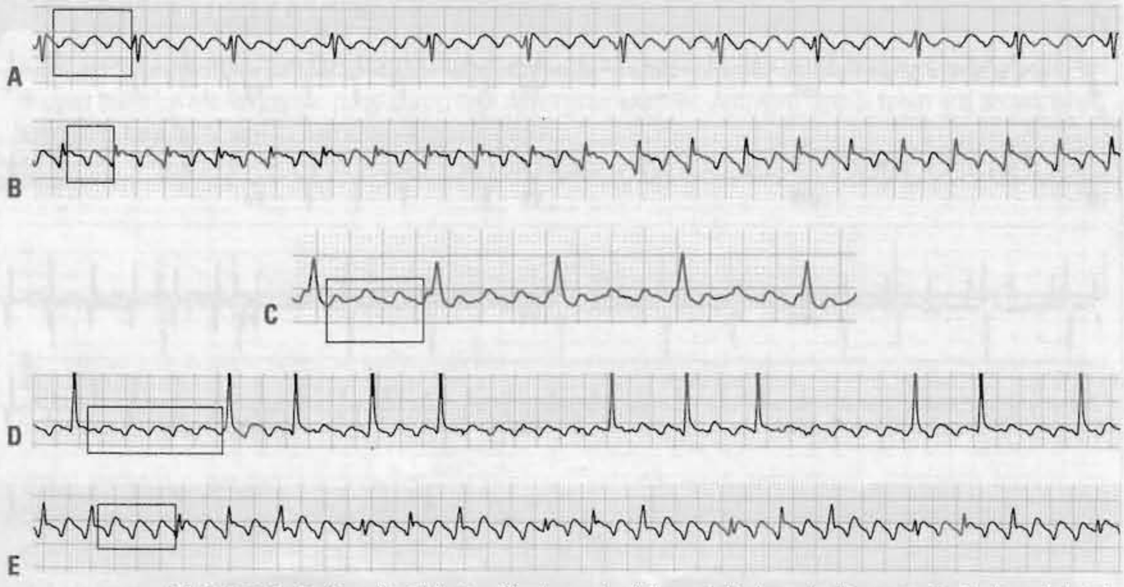


FIGURE 3.3 Atrial flutter. The boxes in A through E show the F waves. **A, C:** show 4:1 atrioventricular conduction. **B:** shows 2:1 atrioventricular conduction. **D, E:** show variable atrioventricular conduction.

Atrial and ventricular rates can be described separately. For example, in Figure 3.3C the atrial rate is about 300 bpm and the ventricular rate is about 75 bpm. Measuring the atrial and ventricular rates is a good way to double check the ratio of atrial to AV conduction. In this example the presence of 4:1 conduction is confirmed, as the atrial rate (300 bpm) is four times the ventricular rate (75 bpm). Simple inspection might lead to mistaking this strip for 3:1 flutter as the fourth F wave is merging with the QRS complex and could be missed.

Although the 12 leads of the standard cardiogram have not been discussed in detail yet, it should be noted that the classic pattern of atrial flutter shows F waves in leads II, III, and aVF (known collectively as the "inferior" leads as they electrically view the bottom of the heart). **Figure 3.4** shows two 12-lead examples of atrial flutter. Note that F waves are present in leads II, III, and aVF, but may not be readily apparent in all leads.

Wandering Atrial Pacemaker and Multifocal Atrial Tachycardia

The ECG pattern is similar in wandering atrial pacemaker and multifocal atrial tachycardia; the major obvious difference is rate. In both cases several areas of the atria are serving as pacemakers (thus the pacemaker is said to be wandering or

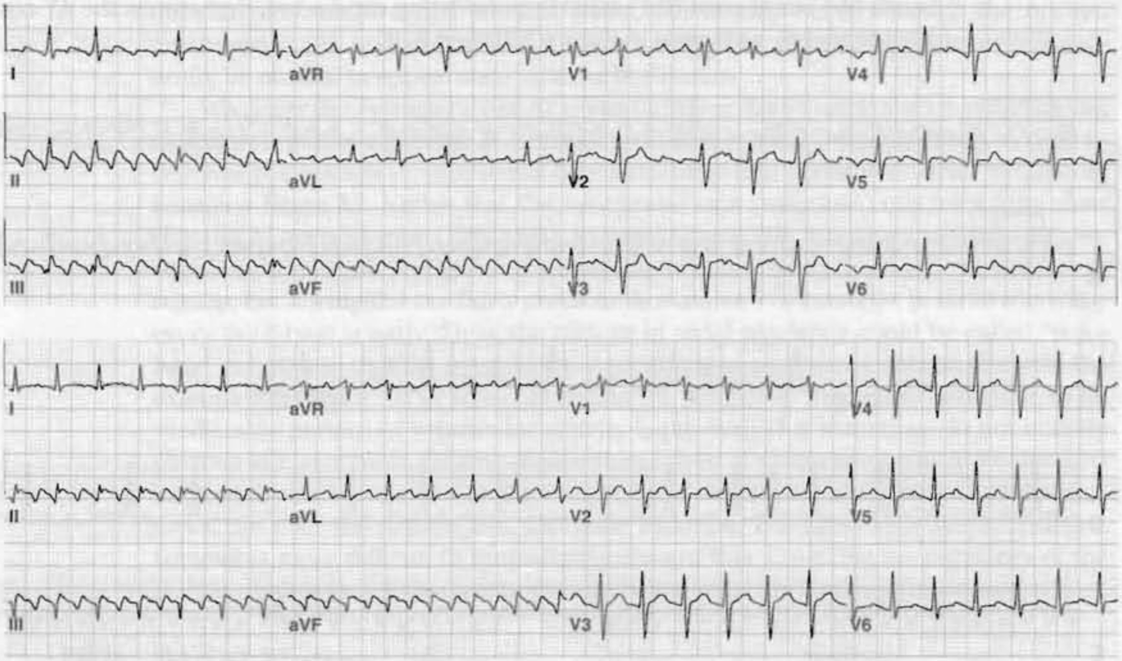


FIGURE 3.4 Atrial flutter. Two examples of a 12-lead ECG are shown here. Note the F waves in leads II, III, and aVF.

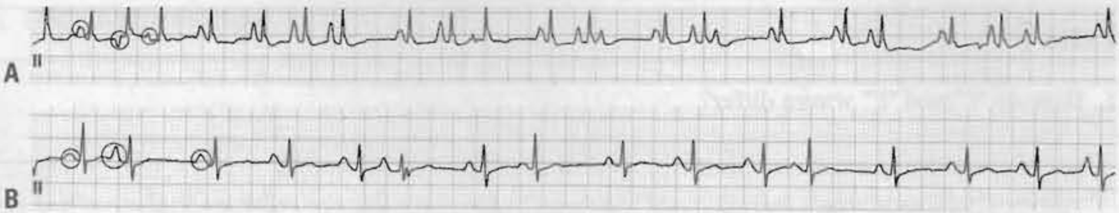


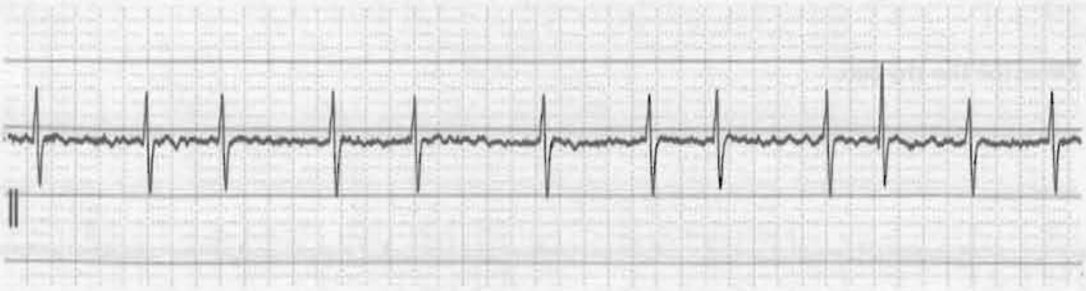
FIGURE 3.5 **A:** Multifocal atrial tachycardia. **B:** Wandering atrial pacemaker. Circles indicate different p wave morphologies.

multifocal). As would be expected in this situation, the P waves differ in appearance. A commonly accepted criterion for these rhythms is the presence of three or more different P wave morphologies. Some of the different P wave shapes are circled in **Figure 3.5**. The PR intervals may also vary, for the same reasons described previously for premature atrial complexes (PAC). In fact these rhythms could be thought of as special cases of "frequent PACs."

Due to the multiple foci the rhythm tends to be irregular, perhaps even "irregularly" irregular. In contrast to atrial fibrillation, P waves are consistently seen in these rhythms. In keeping with general terminology, a rate >100 bpm is usually considered a tachycardia. Thus, Figure 3.5A would be called multifocal atrial tachycardia, whereas Figure 3.5B would be considered a wandering atrial pacemaker (because the rate is <100 bpm).

CLINICAL VIGNETTE REVISITED

A patient in phase III cardiac rehabilitation who previously has had a regular pulse describes feeling a "lot of skipped beats" while taking her pulse during cool down after exercise. A rhythm strip is taken and shows what appears to be a lot of artifact and a very irregular rhythm.



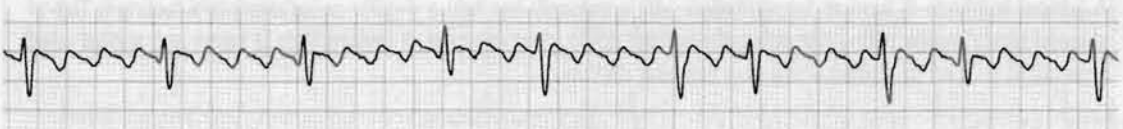
The rhythm strip shows atrial fibrillation; because the patient had previously been in normal sinus rhythm this is an important change in clinical status. Blood pressure was 136/82 mm Hg, which was typical for this patient, and she was in no distress. She was asked to stay in the rehab center while her physician was contacted for instructions.

Quiz 3

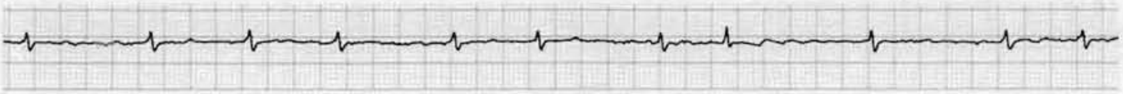
1. How do "f" and "F" waves differ?

2. Describe the regularity of the R-R Intervals in the following arrhythmias:
   - Atrial flutter
   - Wandering atrial pacemaker
   - Atrial fibrillation

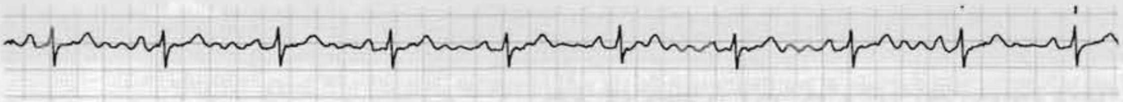
3. Describe the rhythm.

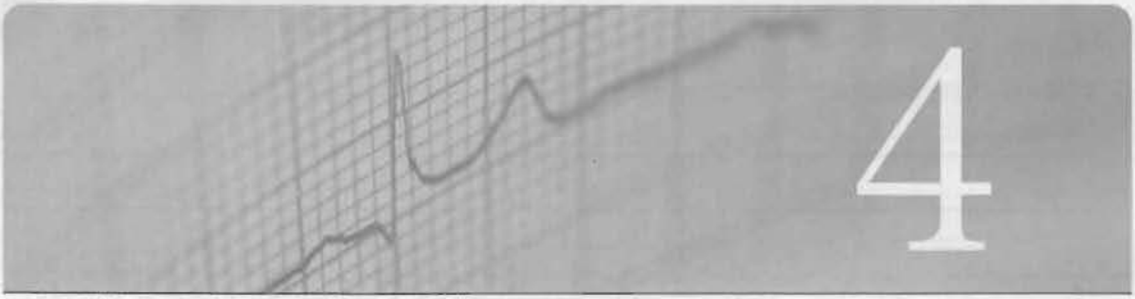


4. Describe the rhythm.



5. Describe the rhythm.





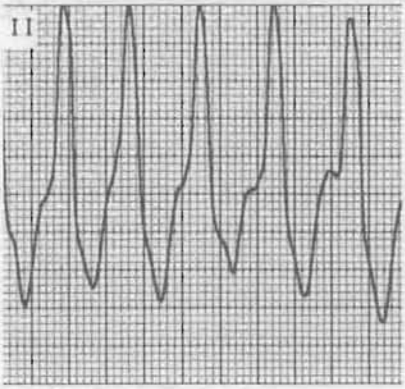
4

VENTRICULAR RHYTHMS

- Premature Ventricular Complexes
- Uniform and Multiform Premature Ventricular Complexes
- Compensatory Pauses and Interpolated Premature Ventricular Complexes
- Ventricular Bigeminy, Trigeminy, and Quadrigeminy
- Ventricular Tachycardia
- Torsade de Pointes
- R on T Premature Ventricular Complexes
- Accelerated Idioventricular Rhythm
- Idioventricular Escape
- Ventricular Fibrillation
- Agonal Rhythms
- Asystole

CLINICAL VIGNETTE

A man in his 30s has reported feeling unwell after exercise. An ECG performed at rest showed normal sinus rhythm, but during recovery from an exercise stress test this occurred:



In previous chapters, it was assumed that the QRS complex would have a normal (narrow) appearance. This assumption was based on the reasoning that conduction in the ventricles would be essentially normal. Various abnormalities in the atria and at or near the atrioventricular (AV) junction have been discussed; however, it was always assumed that depolarizations would reach the ventricular myocardium via the normal conduction system (AV node, bundle of His, bundle branches, and Purkinje fibers). Thus, the QRS complex would appear normal. For example, even during atrial fibrillation, wherein the atrial myocardium is electrically unstable and functionally useless, the depolarizations that did reach the ventricles were assumed to result in normal QRS complexes.

Now imagine a situation where a depolarization has originated in some area of the ventricles. As the ventricular myocardial cells are electrically connected via gap junctions, this depolarization will spread throughout the ventricles; however, since these currents are not spreading through the normal conduction pathways, they will spread more slowly and in a less organized fashion. A QRS complex resulting from currents that spread via these mechanisms has an appearance that is often described as "wide and bizarre"; wide (usually defined as >100 ms) because the spread of current is slower than normal, and bizarre (as compared to the normal QRS morphology) because the electrical activity is not spreading via the normal routes **(Fig. 4.1)**.

KSA Premature Ventricular Complexes

Examine the QRS complexes in **Figure 4.2A**. The QRS in the box is wider and different in appearance from the others. Several different terms including premature ventricular complex (PVC), ventricular premature depolarization (VPD), and ventricular extrasystoles (VES) are used interchangeably to describe these types of events. Here the most common term, PVC, is used. It is an apt term as these beats are early (premature) and appear to arise in the ventricles (ventricular complexes).

By definition, PVCs must be early. Notice in Figure 4.2A that the normal R-R interval is about 640 ms. The PVC (enclosed in the box) is early; the R-R interval from the previous normal beat to the PVC is only 480 ms. Notice also that the PVC is "wide" (QRS duration >100 ms) and has a different morphology than the other QRS complexes. It is also common with PVCs for the QRS complexes and the T waves to be oriented in opposite directions. In this example the QRS complex is positive, whereas the T wave is negative.

KSA Uniform and Multiform Premature Ventricular Complexes

Uniform PVCs look like one another. Three PVCs of similar appearance (uniform) are shown in Figure 4.2B. Figure 4.2D shows two PVCs with different appearances in the same lead; these are called multiform PVCs. Figure 4.2C shows one PVC as seen in three different ECG leads. This illustrates that a given PVC will

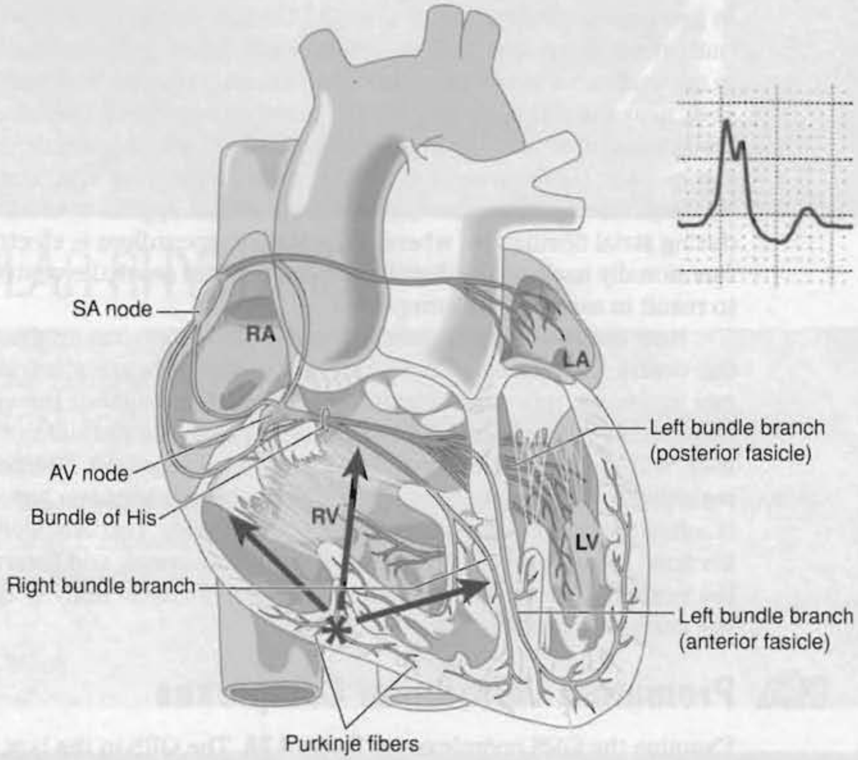

SA node
RA
LA
AV node
Bundle of His
RV
LV
Right bundle branch
Left bundle branch (posterior fasicle)
Left bundle branch (anterior fasicle)
Purkinje fibers

FIGURE 4.1 Ventricular ectopic focus. * indicates the ectopic focus. The corresponding ECG is shown at the top right of the figure. SA, sinoatrial; AV, atrioventricular; RA, right atrium; LA, left atrium; RV, right ventricle; LV, left ventricle.

look different when viewed in different ECG leads; multiform refers to PVCs that look different from each other in the *same* ECG lead.

Uniform PVCs look the same because they arise from the same ectopic focus; therefore, they are sometimes called unifocal PVCs. Multiform PVCs may arise from different areas of the ventricle, so they are sometimes called multifocal. This may not be an appropriate term as it has been found that PVCs that differ in appearance (in the same lead) may actually arise from the same ectopic focus. Thus, the term multiform is preferred, as it does not imply multiple ectopic foci.

KSA Compensatory Pauses and Interpolated Premature Ventricular Complexes

Compare the PVCs in **Figure 4.3**. In Figure 4.3A the PVC is followed by a pause, and then the next P-QRS-T comes when it would have normally occurred had the PVC not intervened. This is an example of a PVC with a *compensatory pause*. The pause

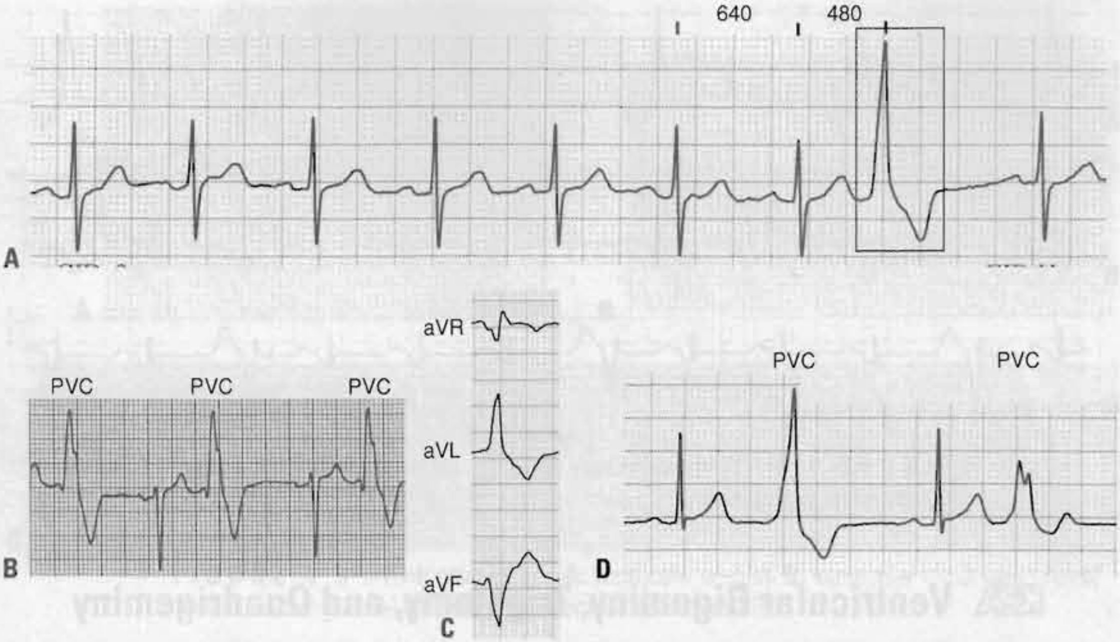


FIGURE 4.2 Premature ventricular complexes (PVCs). **A:** Indicates R-R intervals (ms). **B:** Shows uniform PVCs. **C:** Shows same PVC in three different leads. **D:** Shows multiform PVCs.

after the PVC results in the next R wave coming "on time," in other words, two normal R-R intervals after the last normal R wave. Now examine the PVC in Figure 4.3B. In this case the PVC falls right in the middle of a normal R-R interval and has no effect on the regularity of the other beats (no compensatory pause). This is called an *interpolated PVC.* Some interpolated PVCs do subtly alter the rhythm, but the change is much less pronounced than a compensatory pause.

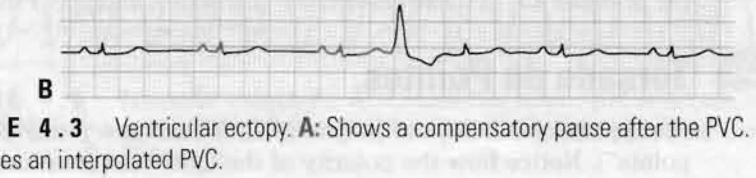


FIGURE 4.3 Ventricular ectopy. **A:** Shows a compensatory pause after the PVC. **B:** Indicates an interpolated PVC.

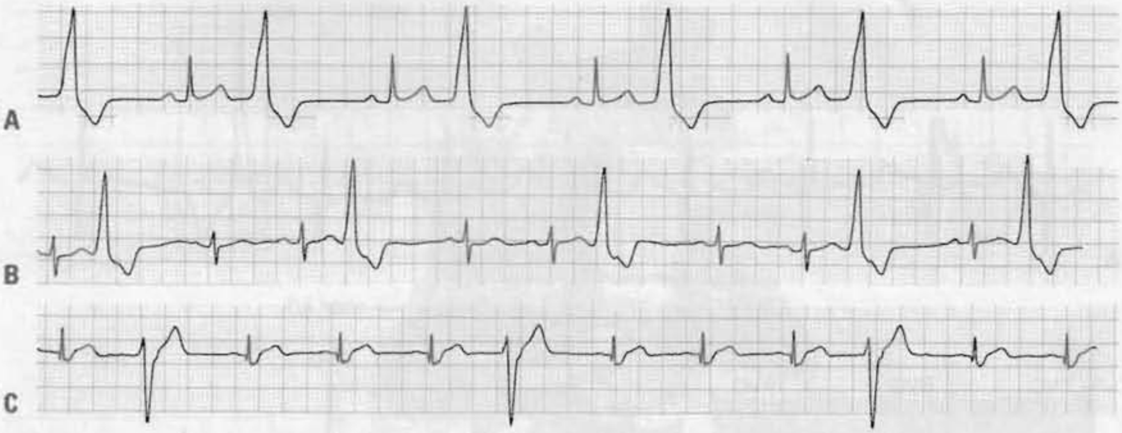


FIGURE 4.4 Ventricular ectopy. **A:** Ventricular bigeminy. **B:** Ventricular trigeminy. **C:** Ventricular quadrigeminy.

Ventricular Bigeminy, Trigeminy, and Quadrigeminy

Patterns of PVCs can be described using similar terminology to that used for premature atrial complexes **(Fig. 4.4)**. Ventricular bigeminy (Fig. 4.4A) is said to exist when every second beat is a PVC, ventricular trigeminy (Fig. 4.4B) when every third beat is a PVC, and ventricular quadrigeminy (Fig. 4.4C) when every fourth is a PVC.

Ventricular Tachycardia

When two PVCs occurs sequentially it is called a *ventricular couplet* and three can be called a *ventricular triplet*. By definition three or more consecutive PVCs is also called *ventricular tachycardia* (V-Tach). Examples of a couplet, a triplet, and some "short runs" of V-Tach are shown in **Figure 4.5**. V-Tach is simply a string of PVCs (three or more). Runs of V-Tach lasting less than 30 seconds are usually described as "short" or "nonsustained," whereas V-Tach lasting more than 30 seconds is usually described as "sustained" V-Tach. The term *self-terminating* is also used to describe short runs of V-Tach.

V-Tach can be further described in terms of the morphology of the QRS complexes. For example, the pattern in **Figure 4.6B,C** could be described as monomorphic V-Tach, as a relatively consistent pattern of QRS complexes is seen. Contrast this with the short run of polymorphic V-Tach shown in Figure 4.6A and the example in **Figure 4.7**; in these cases the PVCs do not all appear the same.

Torsade de Pointes

An important subclass of polymorphic V-Tach is *torsade de pointes* ("twisting of points"). Notice how the polarity of the QRS complexes in Figure 4.7 repetitively

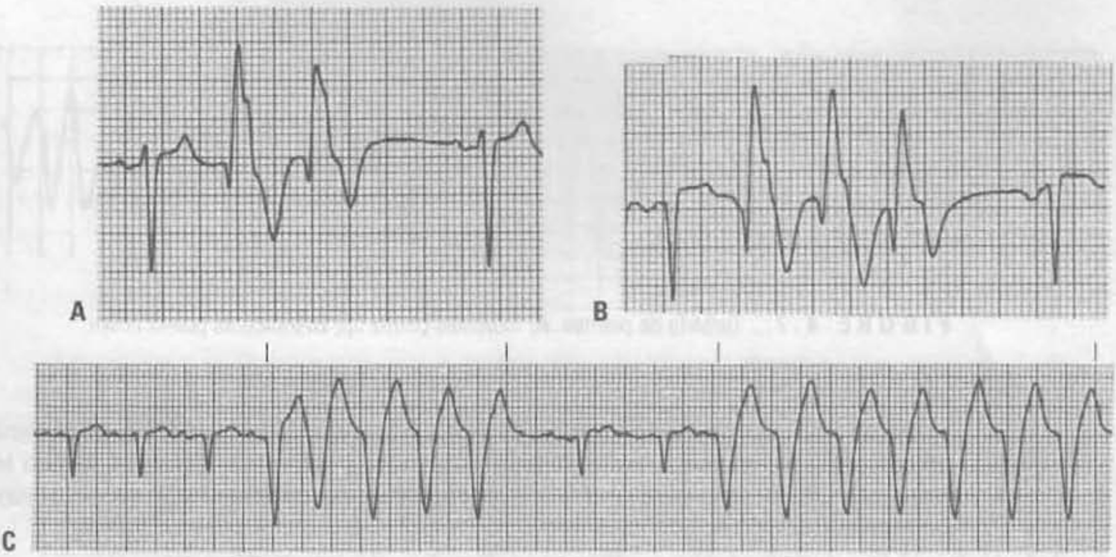


FIGURE 4.5 Ventricular ectopy. **A:** Ventricular couplet. **B:** Ventricular triplet (short run of ventricular tachycardia). **C:** Two runs of ventricular tachycardia.

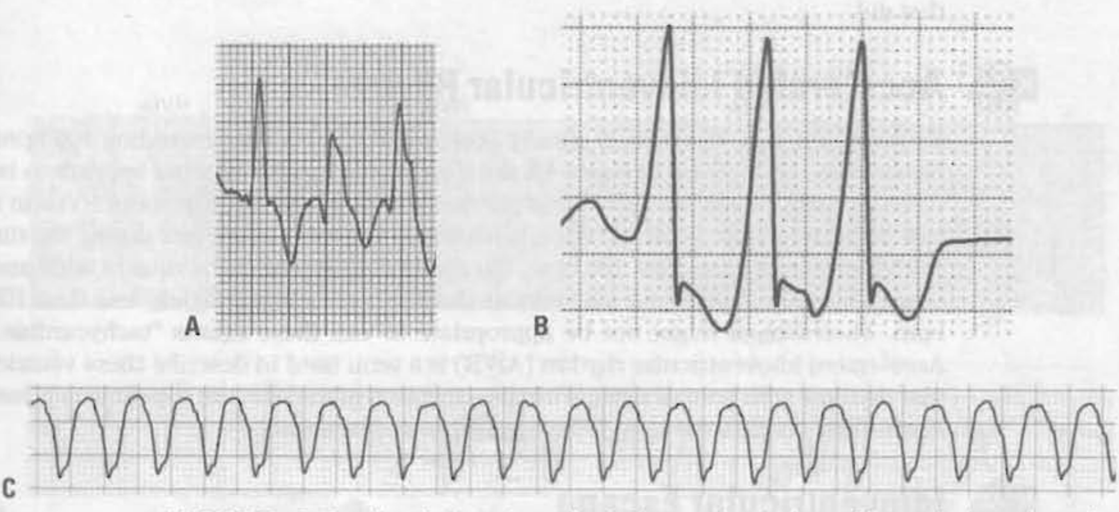


FIGURE 4.6 Ventricular tachycardia. **A:** Nonsustained polymorphic ventricular tachycardia (triplet). **B:** Nonsustained monomorphic ventricular tachycardia (triplet). **C:** Sustained monomorphic ventricular tachycardia.

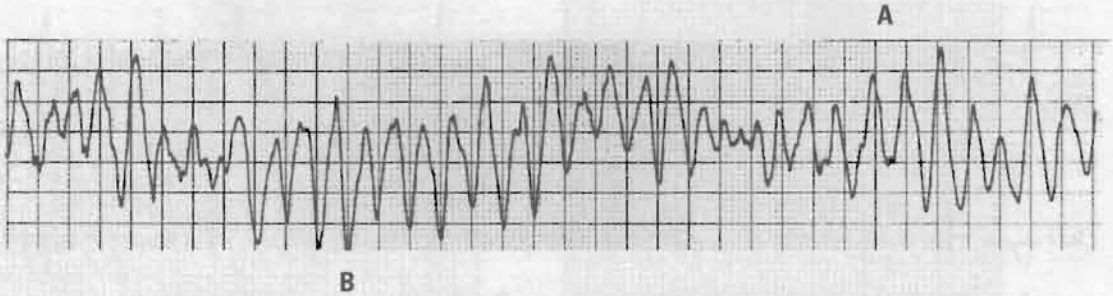


FIGURE 4.7 Torsade de pointes. **A:** Indicates points up. **B:** Indicates points down.

shifts, as evidenced by the "point" of the complexes alternately pointing downward and upward. Recognition of torsade de points as a specific subtype of V-Tach is important, as the cause (electrolyte abnormalities, certain medications, etc.) may be reversible.

R on T Premature Ventricular Complexes

When a PVC falls on the T wave of the preceding beat it is much more likely to lead to a serious arrhythmia such as ventricular fibrillation or V-Tach. This is not to say that these so-called R on T PVCs always lead to dangerous arrhythmias; most often they do not. However, there is a much greater statistical likelihood of an R on T PVC initiating a serious arrhythmia as compared to a non-R on T PVC. **Figure 4.8A** shows an R on T PVC that did not initiate any further arrhythmias; Figure 4.8B shows one that did.

Accelerated Idioventricular Rhythm

By definition, a tachycardia is usually considered to be a rate exceeding 100 bpm. Notice that the rhythms in **Figure 4.9** show several examples of what appears to be three or more consecutive PVCs. As previously described, three or more PVCs in a row is, by definition, V-Tach. Notice, however, in Figure 4.9B the rate during the run of PVCs is clearly less than 100 bpm. Careful measurement of the runs of wide and bizarre beats in Figure 4.9A also reveals that the rate is often slightly less than 100 bpm. Therefore, it might not be appropriate to call these events "tachycardias." Accelerated idioventricular rhythm (AIVR) is a term used to describe these ventricular rhythms with normal rates. With the exception of the rate, the appearance is just like V-Tach, so the term *slow V-Tach* is also sometimes used.

Idioventricular Escape

A ventricular escape (also known as idioventricular escape) rhythm is usually seen in situations where higher pacemakers (sinoatrial [SA] node, AV node, etc.) have

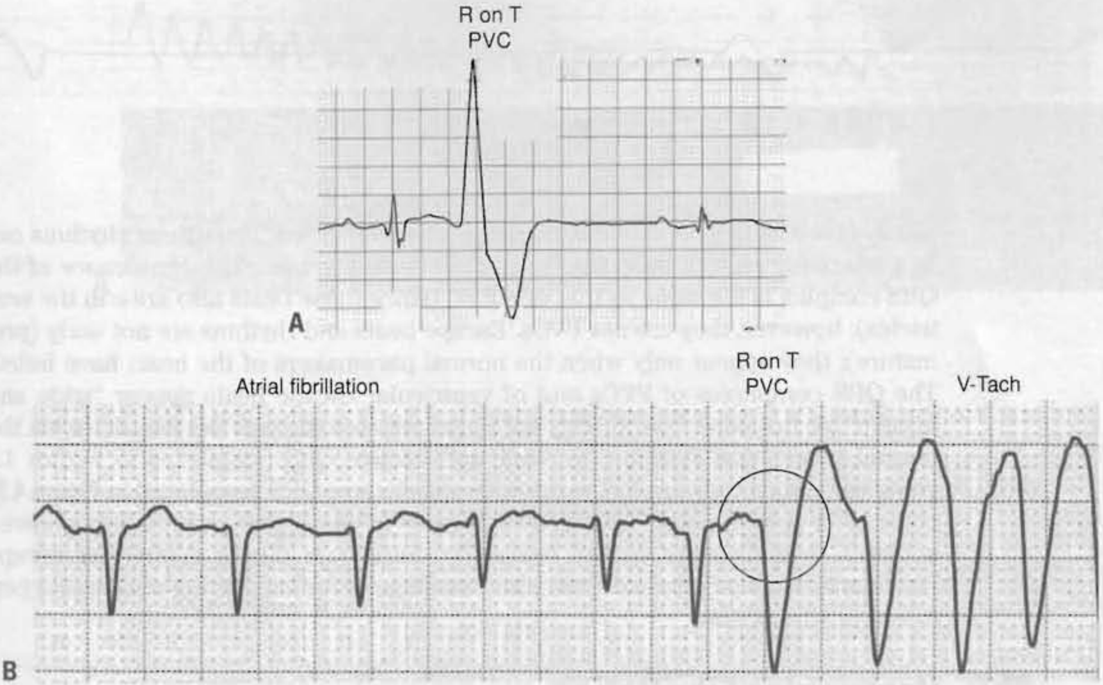

R on T
PVC
A
Atrial fibrillation
R on T
PVC
V-Tach
B

FIGURE 4.8 R on T premature ventricular complex (PVC). V-Tach, ventricular tachycardia. **A:** R on T PVC that does not cause further ectopy. **B:** R on T PVC (circled) initiating V-Tach.

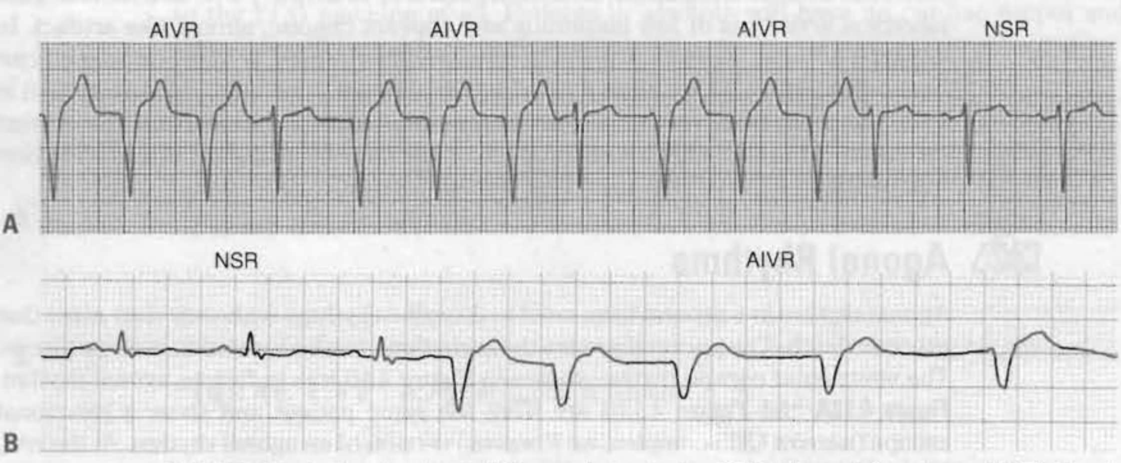

AIVR
AIVR
AIVR
NSR
A
NSR
AIVR
B

FIGURE 4.9 Accelerated idioventricular rhythm (AIVR). Normal sinus rhythm (NSR). **A** and **B** are two examples of AIVR.

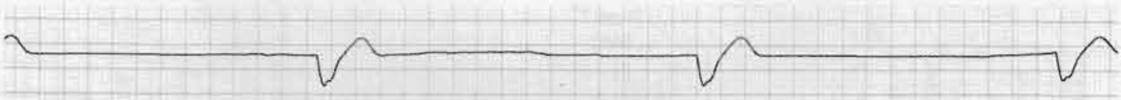


FIGURE 4.10 Idioventricular escape rhythm.

totally failed, or at least failed to depolarize the ventricles. Thus, these rhythms can be protective; without them the heart would cease to beat. The appearance of the QRS complex is the same as that of a PVC (since these beats also arise in the ventricles); however, they are not PVCs. Escape beats and rhythms are not early (premature); they appear only when the normal pacemakers of the heart have failed. The QRS complexes of PVCs and of ventricular escape beats appear "wide and bizarre" for the same reason; they are not conducted through the ventricles via the normal conduction system. The wide and bizarre QRS complexes in Figure 4.2 come early, thus they are PVCs; the wide and bizarre QRS complexes in **Figure 4.10** come after a long pause (during which none of the higher pacemakers showed activity), thus they are escape beats. The heart rate during ventricular escape rhythms is usually quite slow and often inadequate for supporting reasonable perfusion of vital organs.

KSA Ventricular Fibrillation

When the ventricles become electrically incoherent and numerous areas of the ventricular myocardium are rapidly depolarizing in a disorganized fashion, the heart ceases to function as a pump, and cardiac output drops to immeasurable levels. Examples of ventricular fibrillation (V-Fib) are shown in **Figure 4.11A**. Figure 4.11A shows larger undulations, sometimes called *coarse V-Fib.* The lower strip of Figure 4.11A has smaller undulations sometimes referred to as *fine V-Fib.* In both cases the electrical activity is of low amplitude and appears chaotic, almost like artifact. In any type of V-Fib the patient is pulseless and unresponsive, with essentially no cardiac output. If the ECG shows a pattern that appears to be V-Fib, but the patient is alert and oriented or has a pulse and measurable blood pressure, then the rhythm cannot be V-Fib and what is seen on the monitor must be artifact. It is always important to observe the patient as well as the cardiogram.

KSA Agonal Rhythms

Agonal rhythm is a general term used to describe rhythms with very slow rates that precede death. The pacemakers for these rhythms can be located in various places. The ventricular escape rhythm shown in Figure 4.10 was in fact an agonal rhythm. **Figure 4.12A** and Figure 4.12B are from the same patient and show a junctional escape (narrow QRS complex, no P waves) version of an agonal rhythm. At the end, Figure 4.12B the heart has ceased to beat (asystole).

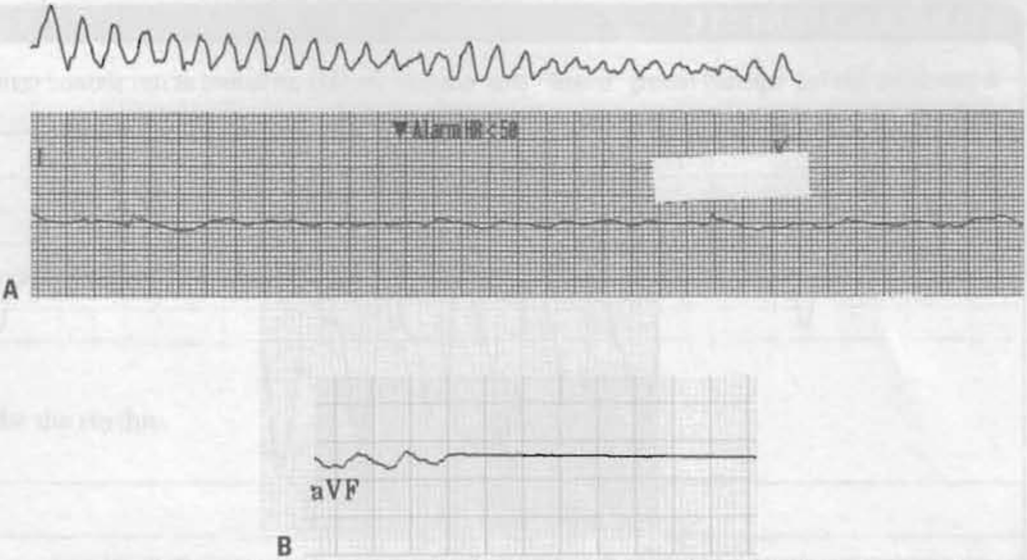


FIGURE 4.11 Ventricular fibrillation (V-Fib). **A:** Shows "coarse" V-Fib (*top*); shows "fine" V-Fib (*bottom*). **B:** Asystole.

Asystole

Asystole, shown at the end of the lower panels of Figures 4.11 and 4.12, is actually the lack of a rhythm; the apparent absence of any electrical activity of the heart. In this situation all myocardial pacemakers have failed and the heart is not depolarizing. The appearance therefore is of a "flat line," as if a patient were not connected to the ECG machine at all. Patients in asystole will have no cardiac output and therefore will be pulseless and unresponsive.

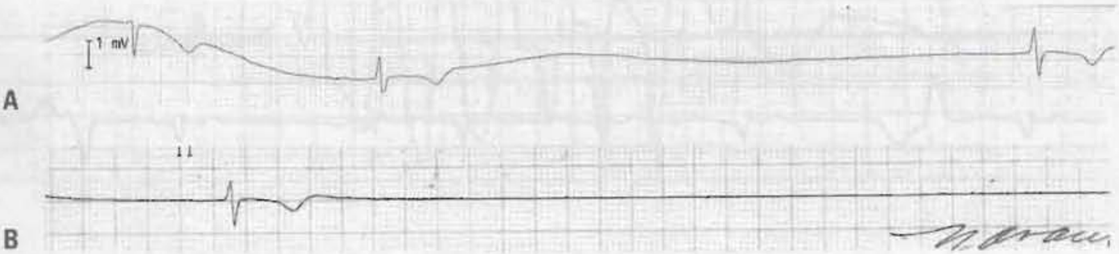
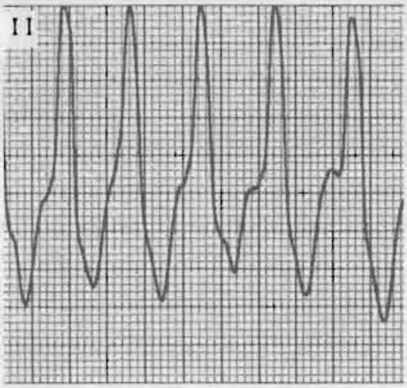


FIGURE 4.12 **A:** Agonal rhythm. **B:** Asystole.

CLINICAL VIGNETTE REVISITED

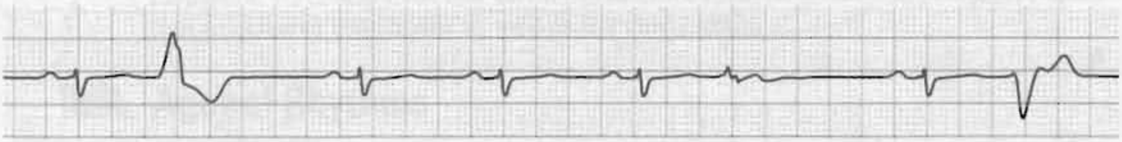
A man in his 30s has reported feeling "unwell" after exercise. An ECG performed at rest showed normal sinus rhythm, but during recovery from an exercise stress test this occurred:



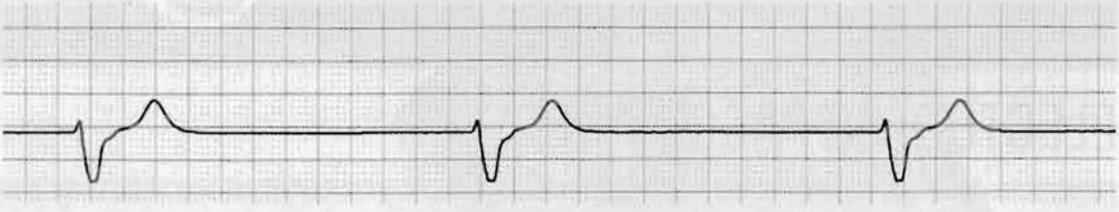
The patient was alert and oriented during the event and it terminated without intervention. Because V-Tach is a serious and potentially life-threatening arrhythmia, the patient was kept in the stress laboratory with the ECG monitored, and the supervising cardiologist was contacted for further instructions.

Quiz 4

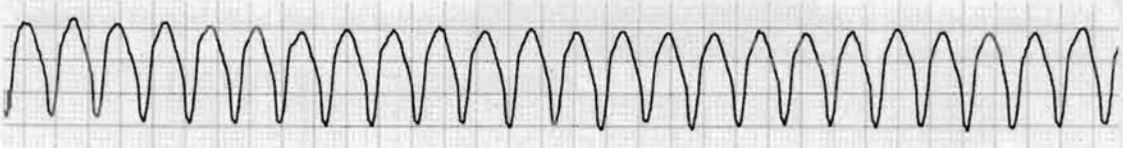
1. Describe the rhythm.



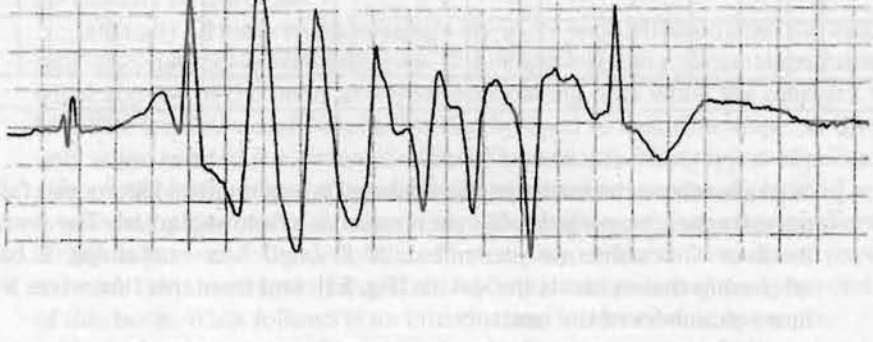
2. Describe the rhythm.

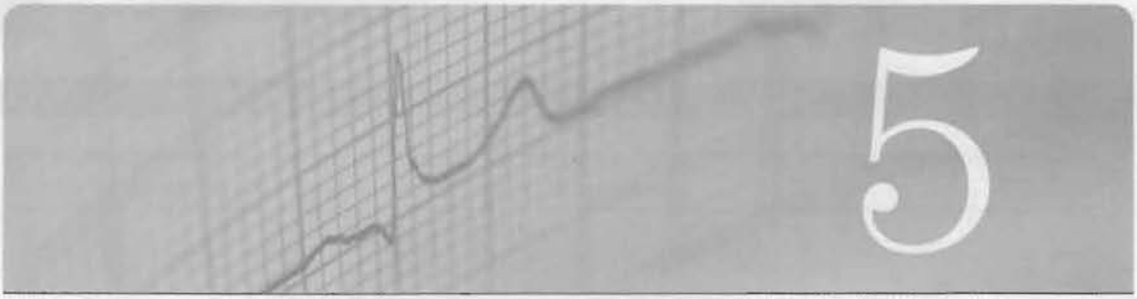


3. Describe the rhythm.



4. Describe the rhythm.





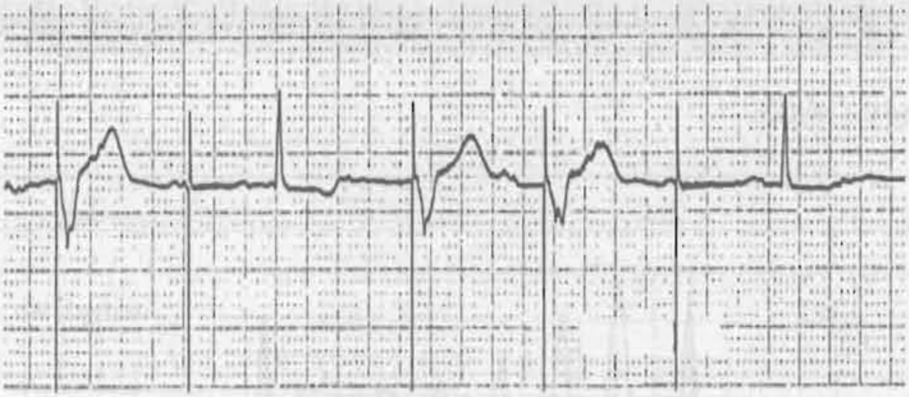
5

ELECTRONIC PACEMAKERS

Ventricular Pacing
Atrial and Dual Chamber Pacing
Pacemaker Malfunctions

CLINICAL VIGNETTE

A longtime participant in phase III cardiac rehabilitation is complaining that she feels easily fatigued by exercise lately. A rhythm strip is run using the site's defibrillator:



In cases where the intrinsic pacemakers (e.g., sinoatrial [SA] node) fail to properly pace the heart, an electronic pacemaker is often implanted. The usual procedure involves subcutaneous placement of a small box containing a battery and a microchip that controls the device **(Fig. 5.1)**, and from this box wires lead to one or more chambers of the heart.

Early transvenous electronic pacemakers were simple devices, which continuously paced at a set rate. The first units used a pacing electrode that was attached by wire to the controlling box and implanted on the epicardial surface. Over time the endocardium of the right ventricle came to be the favored pacing site. This was largely for practical reasons because pacing electrodes in the left

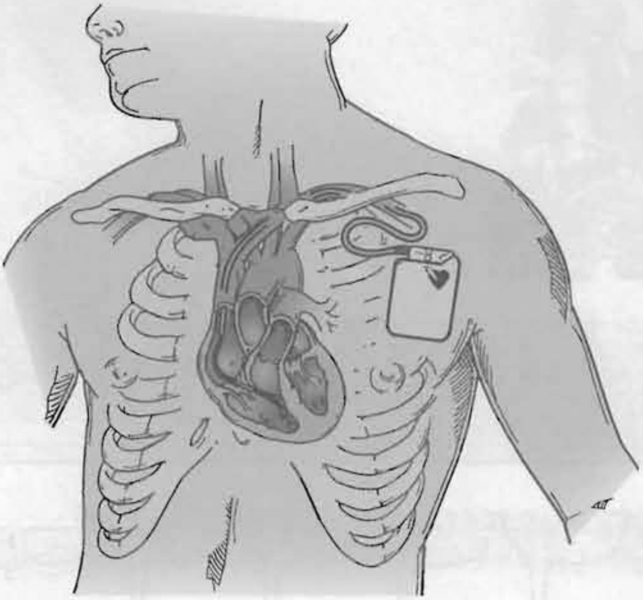


FIGURE 5.1 Electronic pacemaker.

ventricle would cause blood clots to form. Impulses generated in the pacemaker traveled down the wire and depolarized the area of the ventricle near the electrode. This depolarization then spread throughout the ventricles via the electrical connections between the cells (gap junctions). As the depolarization did not spread via the normal conduction pathways, the ensuing QRS complexes were "wide and bizarre." The rate was constant (e.g., 72 beats per minute [bpm]), and the pacemaker functioned monotonously with no regard for the intrinsic electrical activity of the heart.

Virtually all modern pacemakers are more sophisticated than the original "fixed rate" pacemakers described above. For example, newer pacemakers sense the electrical activity of the heart and take over pacing only when the inherent rate is too low. The term *demand pacemaker* can be used to describe these, as they function only when needed (i.e., when a demand is present). Many types of pacemakers are also capable of increasing heart rate in response to increased physical activity that is sensed by various means such as accelerometers. Some pacemakers have the ability to automatically defibrillate if necessary and/or to "overdrive" pace the heart to break certain arrhythmias. A full description of pacemakers is beyond the scope of this book. What follows is an introduction to some basic concepts.

KSA Ventricular Pacing

Figure 5.2 shows an electronic pacemaker that is pacing the heart by depolarizing the ventricles. The dot in the right ventricle represents the pacemaker electrode.

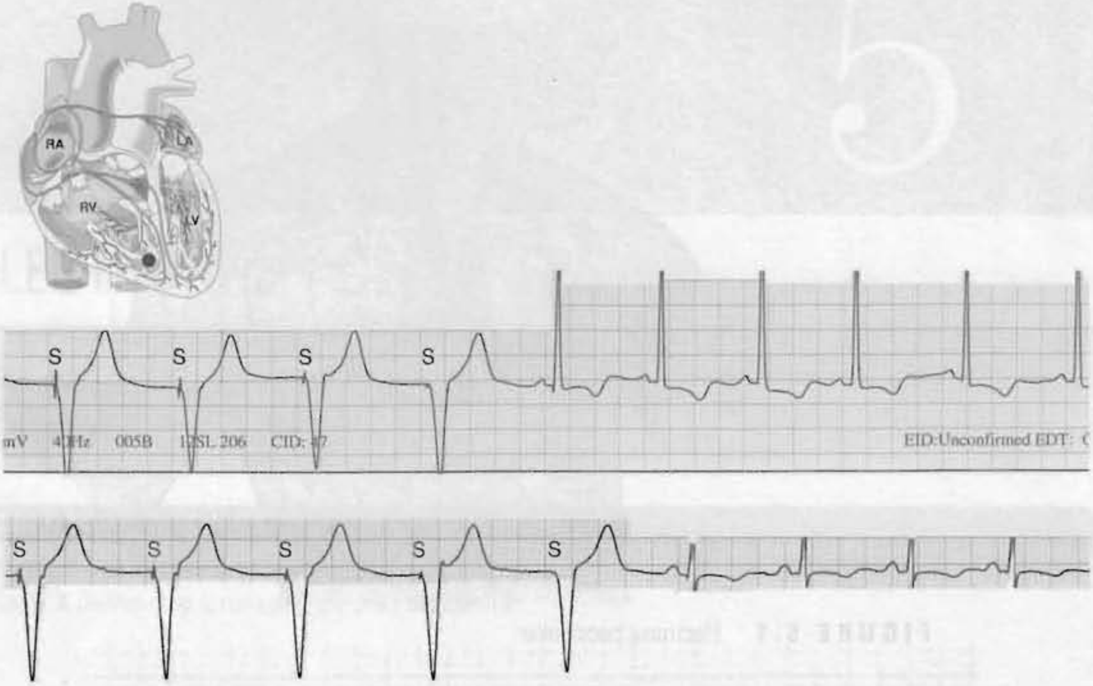


FIGURE 5.2 Ventricular pacing. RA, right atrium; LA, left atrium; RV, right ventricle; LV, left ventricle; S, spike.

This electrode senses the electrical activity of the heart and remains quiescent unless the inherent heart rate drops below a threshold rate programmed into the pacemaker. In the upper ECG strip of Figure 5.2 the first four beats are paced beats, depolarizations initiated by the pacemaker electrode. Each of these four wide and bizarre QRS complexes is preceded by a pacemaker spike (labeled "S"). The heart rate for these paced beats (which can be measured using the normal techniques for rate) is 60 bpm. The fifth QRS complex, and those following it, is not preceded by a pacemaker spike and has a normal (narrow) morphology. This is because the inherent rate has exceeded the pacemaker threshold settings (approximately 60 bpm in this case); the pacemaker's sensor is sensing this, and the pacemaker's pacing electrode is not depolarizing. For these beats the heart is depolarized by the normal inherent mechanisms. The pacemaker only actively paces when the heart's native rate becomes too low.

The ECG in the lower panel of Figure 5.2 is from a different patient. The first five beats are paced (note the pacemaker spikes followed by wide and bizarre QRS complexes); the last four are not (normal QRS complexes preceded by P waves). Again the pacemaker is only pacing the heart when the inherent rate drops below the rate programmed into the pacemaker (again about 60 bpm).

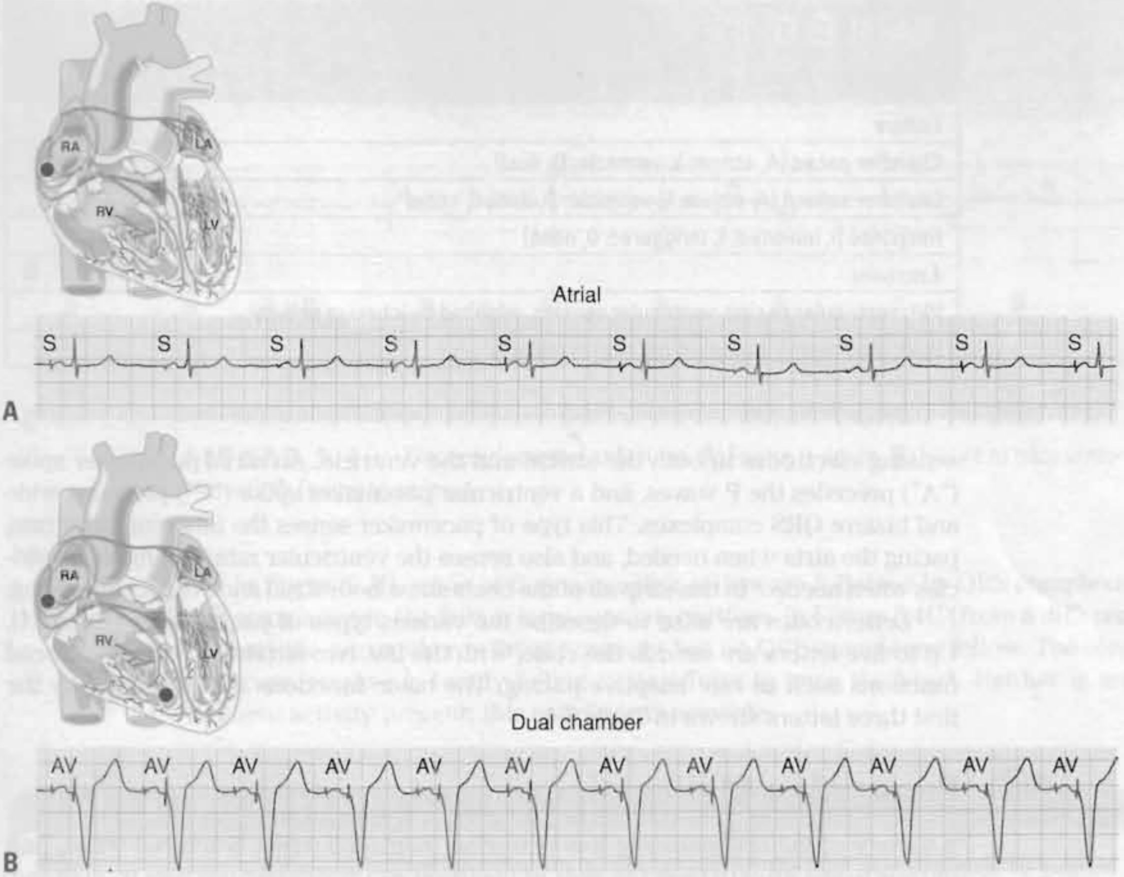


FIGURE 5.3. Electronic pacemakers. **A:** Atrial. **B:** Dual chamber. RA, right atrium; LA, left atrium; RV, right ventricle; LV, left ventricle; S, spike; A, atrial pacemaker spike; V, ventricular pacemaker spike.

Atrial and Dual Chamber Pacing

In **Figure 5.3A** a pacemaker spike ("S") precedes the P wave, not the QRS complex. This is an example of atrial pacing. In this case a sensing and pacing electrode (represented by the dot) is located in the atrium. It senses electrical activity in the atria and fires if the atrial rate drops below the threshold setting of the pacemaker. In this example all of the beats are paced.

Pacemaker spikes followed by a P wave are pacing the atria; spikes followed by a QRS complex are pacing the ventricles. Notice that the QRS complexes in Figure 5.3A are normal in appearance (narrow). Although the pacemaker depolarizes the atria, the current then spreads down into the ventricles via the normal conduction system (atrioventricular [AV] node, bundle of His, etc.). Therefore, the QRS complex is normal in appearance. Figure 5.3B shows a pacemaker with pacing and

TABLE 5.1

Pacemaker Codes

Letters
Chamber paced (A, atrium; V, ventricle; D, dual)
Chamber sensed (A, atrium; V, ventricle; D, dual; 0, none)
Response (I, inhibited; T, 0triggered; 0, none)
Examples
VVI: ventricular pacing, ventricular sensing, inhibited by inherent activity
DDD: dual pacing, dual sensing, dual responses

sensing electrodes in both the atrium and the ventricle. An atrial pacemaker spike ("A") precedes the P waves, and a ventricular pacemaker spike ("V") precedes wide and bizarre QRS complexes. This type of pacemaker senses the inherent atrial rate, pacing the atria when needed, and also senses the ventricular rate, pacing the ventricles when needed. In this strip all of the beats show both atrial and ventricular pacing.

Letter codes are used to describe the various types of pacemakers **(Table 5.1)**. Up to five letters are used in the code, with the last two letters for more advanced functions such as rate adaptive pacing. The basic functions are explained by the first three letters shown in Table 5.1.

Pacemaker Malfunctions

A variety of factors including low batteries, displaced wires, fractured wires, and fibrous tissue growth near the area of insertion can cause pacemakers to malfunction. Two of the major types of malfunction—failure to sense and failure to pace—are described below.

Failure to Sense

Several of the pacemaker spikes in **Figure 5.4A** are inappropriate for a "demand" pacemaker. The second, third, fourth, and fifth spikes come immediately after a native QRS complex. The depolarization of the heart by a native mechanism (likely the SA node, as these QRS complexes are preceded by a small P wave) should have been sensed and the pacemaker should not have fired. The pacemaker is failing at times to sense the inherent activity of the heart. Some of the native QRS complexes in this strip are not followed by pacing spikes; this indicates that the pacemaker is sensing some of the native beats.

Failure to Pace

Figure 5.4B,C shows examples of pacemaker spikes that are not followed by QRS complexes. The pacemaker has fired appropriately but failed to depolarize the heart.

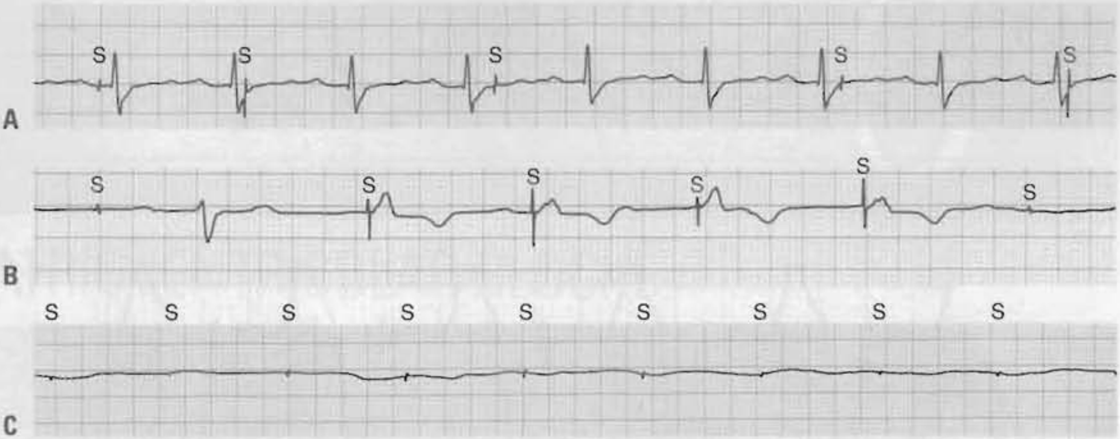
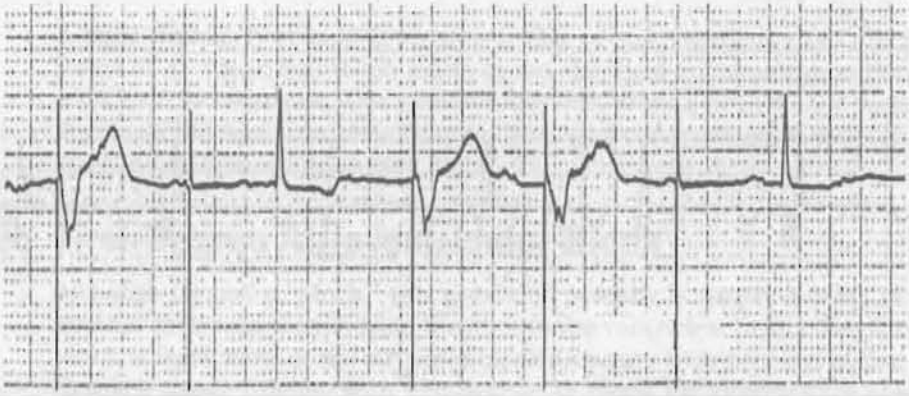


FIGURE 5.4. Electronic pacemaker failures. **A:** Failure to sense. **B:** Failure to pace (intermittent). **C:** Failure to pace.

In Figure 5.4B, some of the pacemaker spikes are followed by QRS complexes and some are not; the failure to pace is intermittent. In Figure 5.4C (from a different patient) the pacemaker is firing regularly, but no QRS complexes follow. The electronic pacemaker is totally failing in its efforts to pace the heart. Neither is any inherent activity present; this patient is in asystole.

CLINICAL VIGNETTE REVISITED

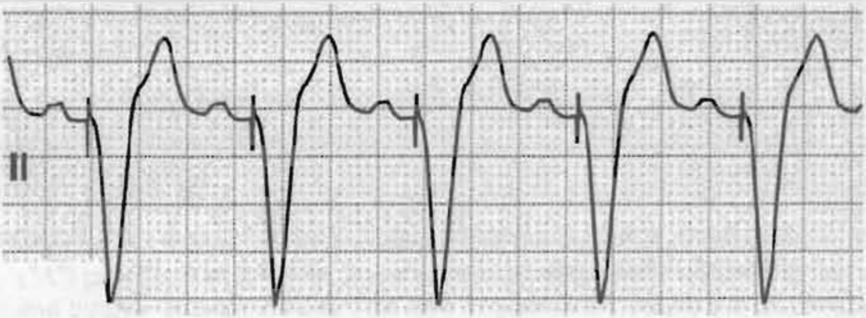
A longtime participant in phase III cardiac rehabilitation is complaining that she feels easily fatigued by exercise lately. A rhythm strip is run using the site's defibrillator:



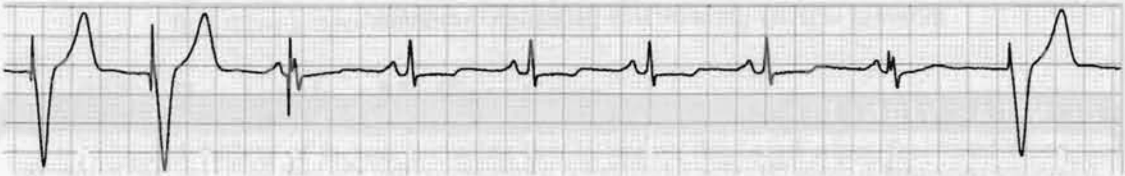
The patient's pacemaker is malfunctioning. Some pacemaker spikes are not followed by QRS complexes, indicating an intermittent failure to pace. She is instructed not to exercise, and her physician is contacted for further instructions.

Quiz 5

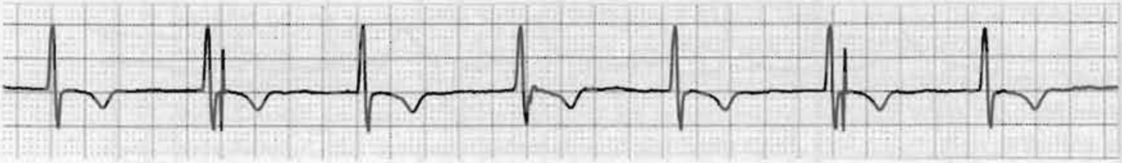
1. Describe the rhythm. Is it possible that this rhythm is from a dual chamber pacemaker?

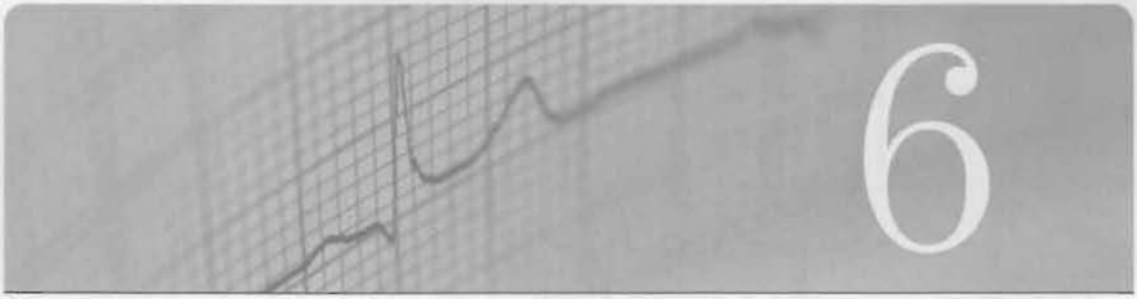


2. Describe the rhythm.



3. Describe the rhythm.





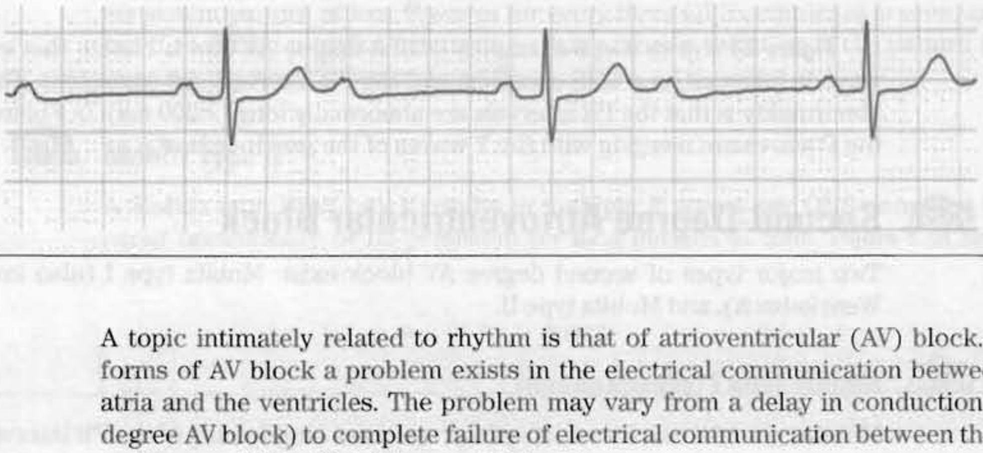
6

ATRIOVENTRICULAR BLOCKS

First-Degree Atrioventricular Block
Second-Degree Atrioventricular Block
Third-Degree Atrioventricular Block
Atrioventricular Dissociation

CLINICAL VIGNETTE

A patient with previously "normal" resting ECGs presents for a treadmill exercise stress test. Before the stress test, a resting ECG is taken.



A topic intimately related to rhythm is that of atrioventricular (AV) block. In all forms of AV block a problem exists in the electrical communication between the atria and the ventricles. The problem may vary from a delay in conduction (first-degree AV block) to complete failure of electrical communication between the atria and the ventricles (third-degree AV block).

First-Degree Atrioventricular Block

Although termed a "block," this condition actually is simply a delay in the conduction of depolarization from the atria to the ventricles. In all other forms of AV block at least some of the atrial depolarizations are truly blocked in that they fail to reach the ventricles. With first-degree AV block PR intervals are consistently long (≥200 ms), but all of the atrial depolarizations do reach the ventricles. If the PR interval is exactly 200 ms the term *borderline* first-degree AV block is sometimes used.

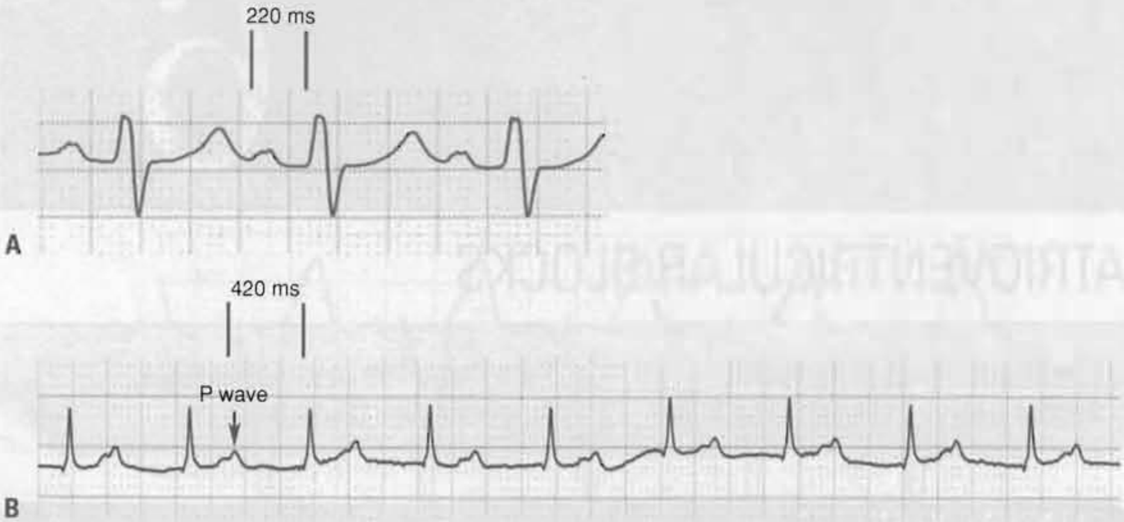


FIGURE 6.1 First-degree atrioventricular block. In (**A**) and (**B**), PR intervals are between the vertical lines.

Figure 6.1 shows some examples of first-degree AV block. Notice that every P wave is followed by a QRS complex, and the PR intervals are consistent. The only abnormality is that the PR intervals are abnormally long (≥200 ms). In Figure 6.1B, the P waves are merging with the T waves of the previous beat.

Second-Degree Atrioventricular Block

Two major types of second degree AV block exist: Mobitz type I (also know as Wenckebach), and Mobitz type II.

Mobitz Type I (Wenckebach)

Mobitz type I AV block consists of a progressive lengthening of the PR interval, culminating in a P wave that does not conduct down into the ventricles and therefore is not followed by a QRS complex. The missing (often referred to as *dropped*) QRS causes a break in the rhythm that leads to a characteristic appearance of groups of P-QRS-Ts with spaces in between. This is sometimes referred to as a *group beating pattern.*

Notice in **Figure 6.2A** how the PR interval becomes longer with each succeeding cycle until a P wave is not followed by a QRS complex. After the space caused by the missing QRS, the pattern begins again. In Figure 6.2B, the groups are shaded. Groups of QRS complexes may appear with other arrhythmias, so the identification of progressively lengthening PR intervals leading to a P wave not followed by a QRS complex is needed to confirm the diagnosis of Wenckebach; however, the appearance of

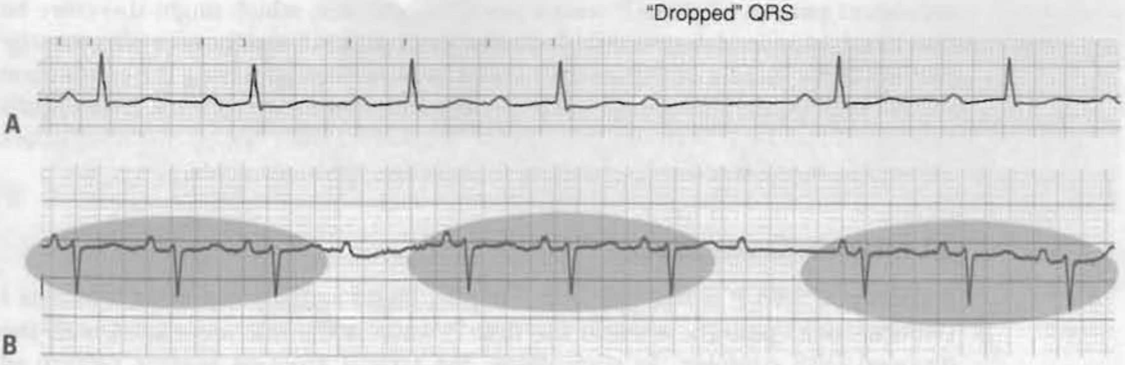


FIGURE 6.2 **A:** Second-degree atrioventricular block. **B:** Shows groups of QRS complexes (shaded).

grouped beating should greatly raise the index of suspicion for second-degree AV block Mobitz type I.

The Wenckebach pattern may be persistent or may occur intermittently. The ratio of P waves to QRS complexes may vary or remain constant. In Figure 6.2B, a consistent pattern of four P waves for every three QRS complexes is seen, and this can be referred to as a 4:3 pattern. In many cases a variation in the pattern occurs (e.g., sometimes 3:2, sometimes 4:3).

Mobitz Type II

A Mobitz type II AV block results in multiple P waves per QRS complex. It may occur occasionally or be persistent for long periods of time. **Figure 6.3A** shows a

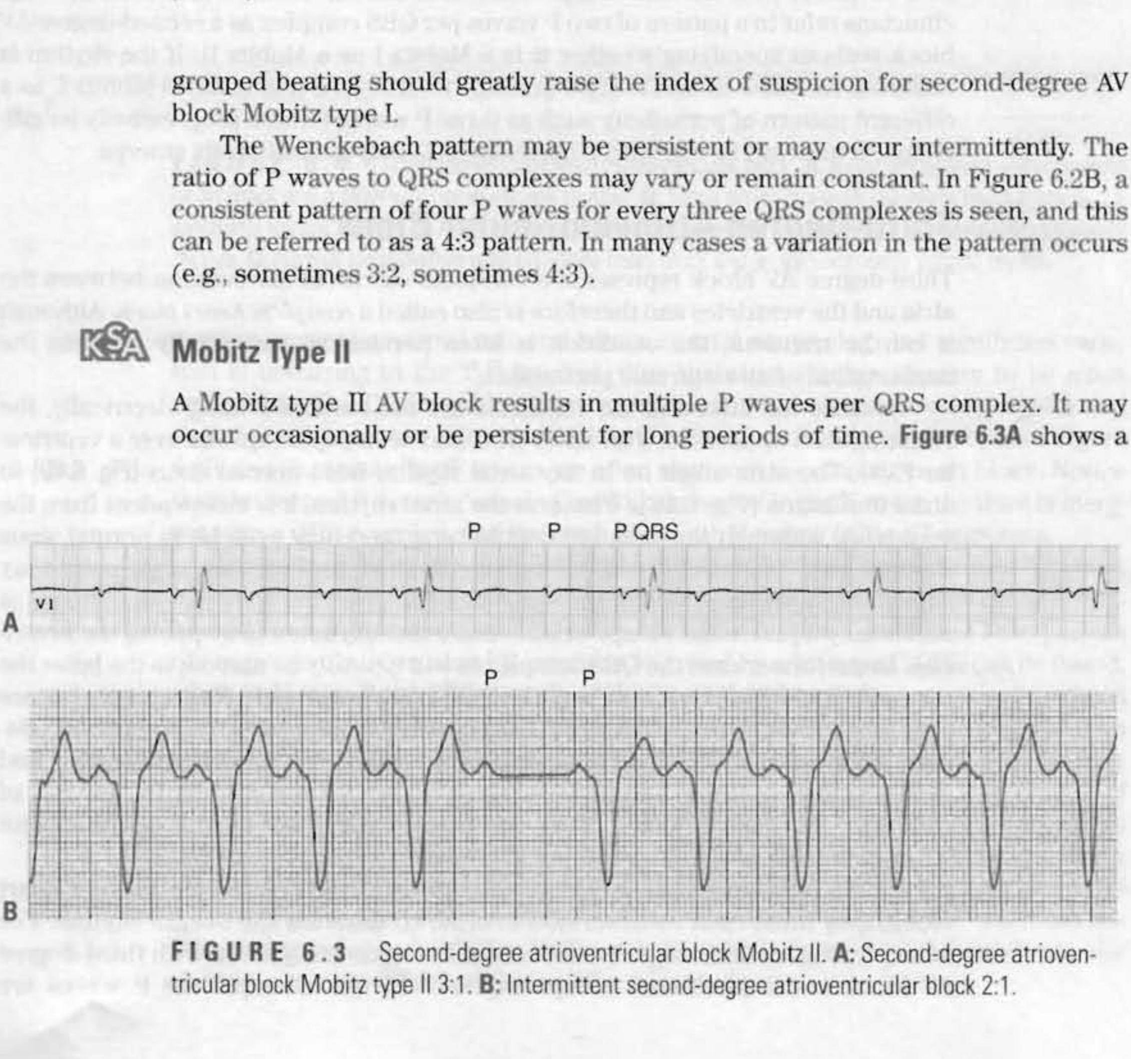


FIGURE 6.3 Second-degree atrioventricular block Mobitz II. **A:** Second-degree atrioventricular block Mobitz type II 3:1. **B:** Intermittent second-degree atrioventricular block 2:1.

consistent pattern of three P waves per QRS complex, which might therefore be described as second-degree AV block Mobitz II with 3:1 conduction; whereas the pattern in the middle of Figure 6.3B could be described as having 2:1 conduction (and is only occurring intermittently). Conduction patterns may vary, for example between 3:1 and 4:1. Notice in these examples that for the P waves that do conduct through to the ventricles the resulting PR intervals are consistent.

2:1 Atrioventricular Block

A pattern of two P waves per QRS complex could actually represent a Mobitz I (Wenckebach) pattern, wherein the first P wave is the one associated with the dropped QRS complex. In such cases, the typical grouped beating pattern of Wenckebach does not occur. A pattern of two P waves per QRS complex could also be a Mobitz II with 2:1 conduction. Often it is difficult to tell which it is, so some clinicians refer to a pattern of two P waves per QRS complex as a second-degree AV block without specifying whether it is a Mobitz I or a Mobitz II. If the rhythm is observed for some time it is often possible to discern if it is in fact a Mobitz I, as a different pattern of periodicity such as three P waves (with a progressively lengthening PR interval) to two QRS complexes (3:2) may intermittently emerge.

Third-Degree Atrioventricular Block

Third-degree AV block represents a complete electrical dissociation between the atria and the ventricles and therefore is also called a *complete heart block*. Although it can be transient, the condition is often permanent and usually requires the implantation of an electronic pacemaker.

Because the atria and the ventricles are not communicating electrically, the resulting ECG appears as if an atrial ECG had been superimposed over a ventricular ECG. The atria might be in any atrial rhythm from normal sinus **(Fig. 6.4D)** to atrial fibrillation (Fig. 6.4C). Whatever the atrial rhythm, it is independent from the ventricles. Although the atria are depolarizing (and may even be in normal sinus rhythm), as these depolarizations are not reaching the ventricles, some subsidiary pacemaker must take over for the ventricles, just as if atria were not depolarizing at all. Usually a junctional escape or idioventricular rhythm will be pacing the ventricles. In the former case the QRS complexes will typically be narrow, in the latter the rate will tend to be very slow and the QRS complexes wide. These concepts are consistent with the escape rhythms discussed in Chapters 2 and 4. In previous discussions of escape rhythms the higher pacemakers (e.g., sinoatrial node) had failed, leading to lower pacemakers taking control of the rhythm. In the case of complete (third-degree) heart block, the higher pacemakers may be operating, but the depolarizations are not reaching the ventricles.

As the atrial and ventricular rhythms are independent in this type of heart block, they must each be described in order to describe the overall rhythm. For example, Figure 6.4B might be described as "sinus arrhythmia with third-degree AV block and a junctional escape rhythm." In this example the P waves are

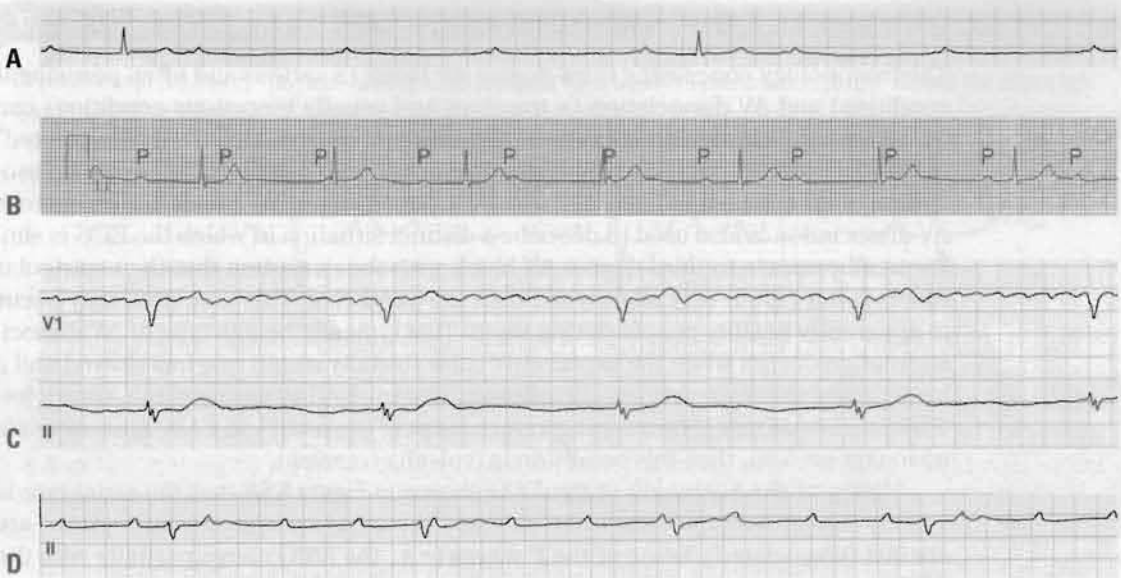


FIGURE 6.4 Third-degree (complete) atrioventricular block. **A:** Sinus bradycardia with complete heart block and a slow junctional escape rhythm. **B:** Sinus arrythmia with complete heart block and a junctional escape rhythm. **C:** Atrial fibrillation with complete heart block and an idioventricular-escape rhythm. **D:** Normal sinus rhythm with complete heart block and an idioventricular escape rhythm.

coming along at a normal rate and have a normal morphology but significant variation is occurring in the P-P interval, thus the atrial rhythm appears to be sinus arrhythmia. The QRS complexes are narrow and the ventricular rate is regular and slightly <50 beats per minute, consistent with a junctional escape rhythm. Figure 6.4B also illustrates that P waves are often obscured in complete heart block. Notice that the second P wave is "missing" and likely buried in the T wave, the third is merging with a QRS complex, and the sixth and ninth P waves fall in ST segments.

In common with complete heart block, both types of second-degree AV block also have more P waves than QRS complexes, but in second-degree block a relationship exists between the atrial and ventricular rhythms. In Mobitz I the pattern of progressively increasing PR intervals followed by a "dropped" QRS can be found, while in Mobitz II the PR intervals of the beats that do conduct are consistent and even if the conduction is varying (e.g., between 2:1 and 3:1) a relationship between atrial and ventricular activity is present. In third-degree AV block a pattern may occasionally appear to resemble something else (e.g., progressive lengthening of the PR interval), but careful inspection will reveal no consistent relationship between the atrial and ventricular rhythms. A long rhythm strip may be helpful in cases where the rhythm is not readily apparent. In true third-degree AV block the atrial rate must also be faster than the ventricular rate. If the atria and ventricles are functioning independently, but the ventricular rate is faster than the atrial rate, the rhythm is termed *AV dissociation* (discussed below).

Atrioventricular Dissociation

The terminology concerning third-degree AV block (a serious and often permanent condition) and AV dissociation (a transient and usually innocuous condition) can be confusing. During both conditions the atria and the ventricles are "dissociated" in that they are functionally electrically independent. Therefore the term AV dissociation often comes up during discussions of third-degree AV block. Unfortunately, AV dissociation is also used to describe a distinct situation in which the ECG is similar in all respects to third-degree AV block with the exception that the ventricular rate is faster (often very slightly so) than the atrial rate. This condition may occur in apparently healthy people during sleep. This typically benign type of AV dissociation often occurs when the supraventricular (usually sinus) rate has slowed and a lower pacemaker (junctional or idioventricular) begins to pace the ventricles. When the atrial rate increases the ventricles are "captured" and AV dissociation is no longer present, thus this condition is typically transient.

Notice at the beginning of the ECG shown in **Figure 6.5A** that the atrial rate is slightly slower than the ventricular rate and the atrial and ventricular rhythms are distinct (dissociated). Some of the P waves (e.g., the fifth) merge partially with the QRS complexes and therefore are partially obscured. Other P waves (e.g., the seventh) are not seen as atrial and ventricular depolarization occurring concurrently, and the QRS is therefore obliterating the P wave. At the end of the strip the atria appear to have "captured" the ventricles. The ventricular rate has slowed, facilitating the return of sinus rhythm.

Figure 6.5B shows another example of AV dissociation. Again, several of the P waves are merging with QRS complexes. In this example the ventricular rate is somewhat irregular and a premature ventricular complex falls in the middle of the strip.

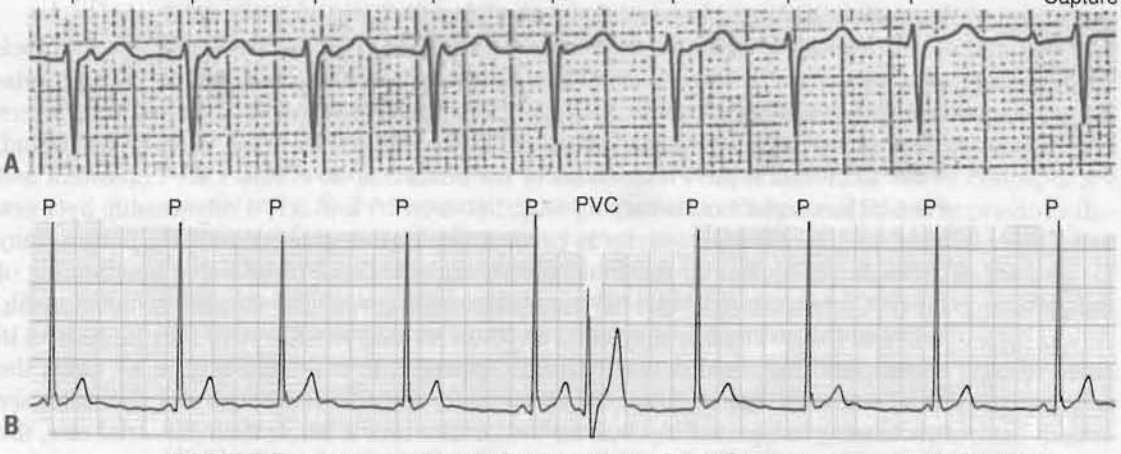
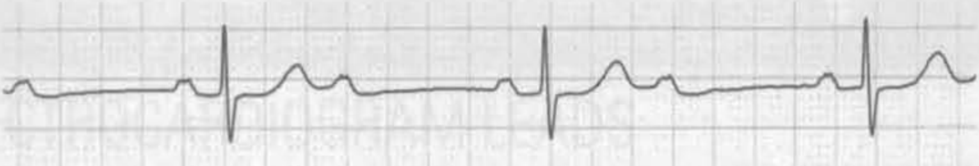


FIGURE 6.5 Atrioventricular dissociation. **A** and **B:** Two examples of AV dissociation.

CLINICAL VIGNETTE REVISITED

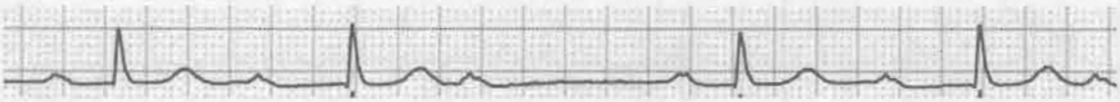
A patient with previously "normal" resting ECGs presents for a treadmill exercise stress test. Before the stress test, a resting ECG is taken.



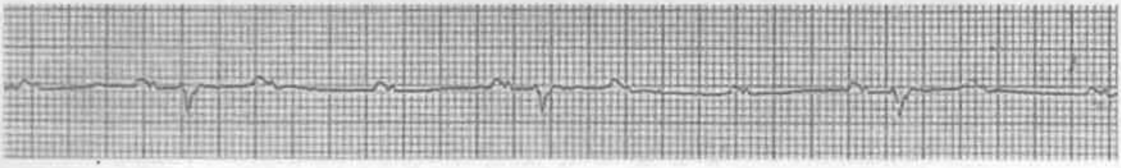
A 2:1 AV block is present. Observation and longer rhythm strips may reveal if it is a Mobitz I or a Mobitz II. Mobitz II is a more serious form of block, but in either case, as the patient previously was reported to be in normal sinus rhythm, this represents a significant change in clinical status. The cardiologist supervising the stress laboratory was informed, and the patient was not placed on the treadmill pending further evaluation.

Quiz 6

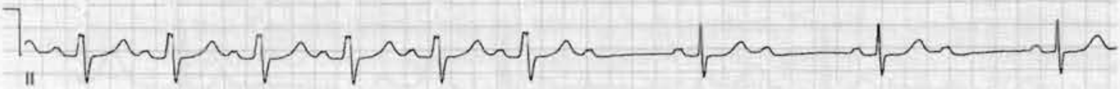
1. Describe the rhythm.

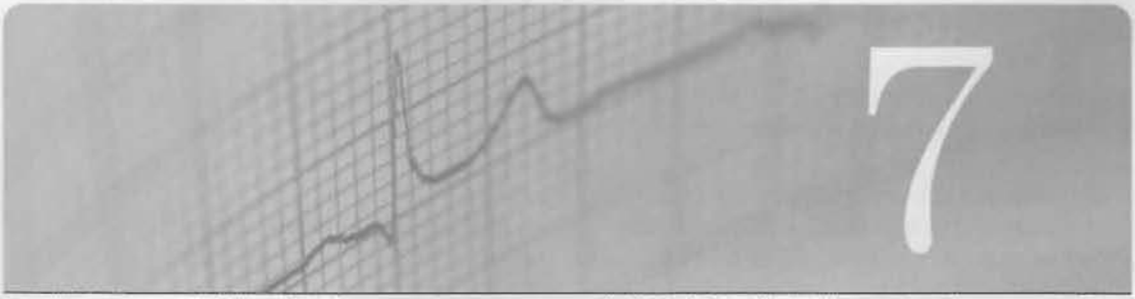


2. Describe the rhythm.



3. Describe the rhythm.





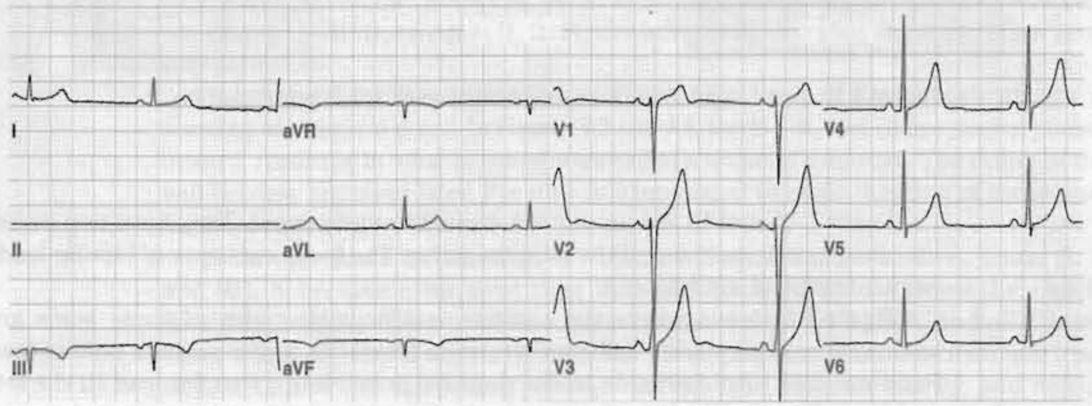
7

ELECTROCARDIOGRAM LEADS

- 12 Standard Leads
- Electrodes
- Mason-Likar Lead Placement
- Localization of Leads
- Normal Patterns

CLINICAL VIGNETTE

A student intern under your supervision is puzzled by a resting ECG and wants to know if "the lead II electrode fell off."



The standard ECG has 12 leads; these may be thought of as different electrical views of the heart. Additional leads (e.g., right-sided) are sometimes used, but for most of the remainder of this book we will discuss the standard leads.

KSA 12 Standard Leads

Limb leads: I, II, III, aVR, aVL, aVF

Chest (precordial) leads: V_1, V_2, V_3, V_4, V_5, V_6

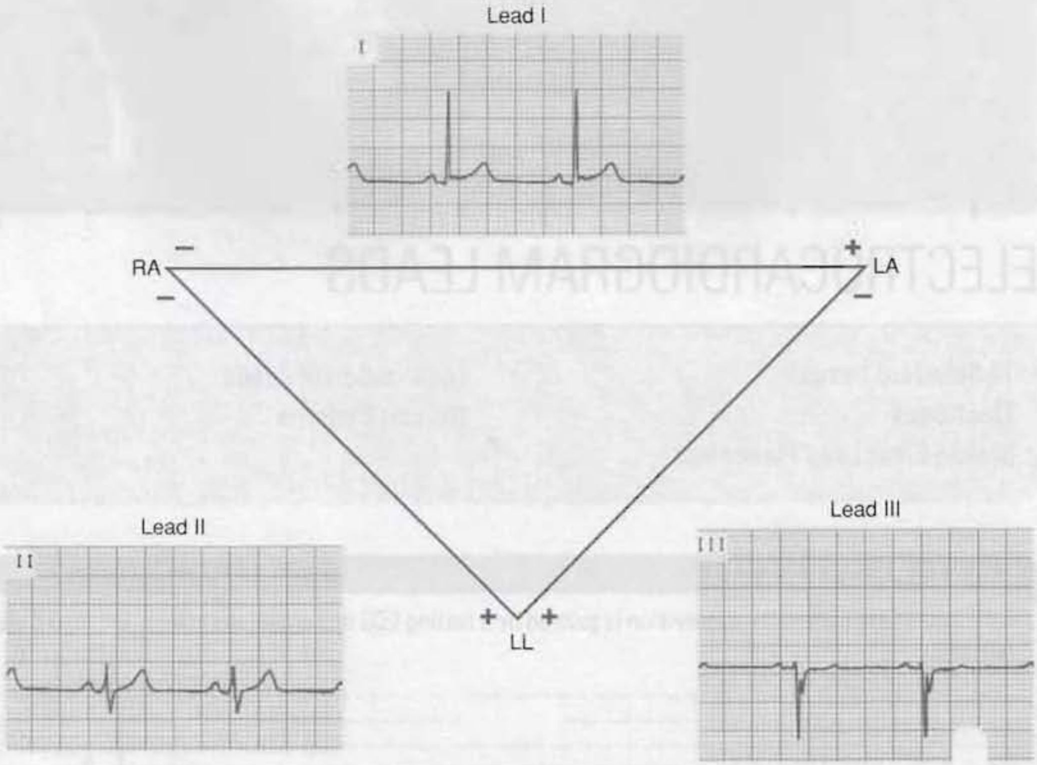


FIGURE 7.1 Einthoven's triangle. RA, right arm; LA, left arm; LL, left leg.

Leads I, II, and III are among the easiest to understand. They have two poles, one designated positive and the other negative. The basic concepts of bipolar leads were introduced in Chapter 2.

Figure 7.1 shows where the positive and negative poles of these leads are located. For example, the left arm electrode functions as the positive pole of lead I and the right arm electrode as the negative pole. Notice that for lead III the left arm electrode functions as the negative pole and the left leg electrode is the positive pole. A given electrode can be made to function as either pole (positive or negative) depending on which lead is being recorded. This also illustrates that electrodes are not leads. Electrodes are the conductive patches attached to the patient. The ECG machine uses the currents detected by these electrodes to obtain the various ECG leads.

Figure 7.1 also shows a triangle formed by leads I, II, and III. Known as Einthoven's triangle, for the physiologist and Nobel Laureate Willem Einthoven who invented the modern ECG machine, this triangle diagrams the relationships between these leads. The three small ECG strips in Figure 7.1 show ECGs recorded simultaneously in these leads. Obviously the same electrical events can appear quite different when viewed in different leads. This is the purpose of the different leads, if the ECG looked the same then multiple leads would not add any information.

Given the orientation of leads I, II, and III, the voltage in lead II at a given point in time should equal that obtained in leads I and III, so in ECG it is sometimes said that lead I + III = II. This has some practical utility as it is one way to verify whether the limb lead wires were placed in the correct positions. For instance, if the P waves in lead II are negative, this may represent an ectopic rhythm, or it may simply indicate misplacement of the wires. If the patient is present one should check that the wires are correctly attached to the electrodes, as it is a common mistake to reverse the right and left side wires (e.g., RA, right arm; LA, left arm). If the patient is not present, one method of verifying proper limb lead wire placement is to ascertain if I + III = II. This is done quite simply; deflections above the baseline are given positive values, while deflections below the baseline are assigned negative values. For instance, a 2-mm deflection below the baseline would be counted as –2 mm (or –0.2 mV), whereas a 2-mm upward deflection would be counted as +2 mm (or +0.2 mV). At a given time point the sum of I + III should equal II. If not, it is likely that the wires were misplaced. In different leads the peak of the R wave or other events may not occur at exactly the same time, so it is important to measure at the same time in the different leads. If the peaks of the R waves are not simultaneous a ruler placed vertically so that it crosses leads I, II, and III at a given time point can assist in measurement. **Figure 7.2** shows an example where I + III does equal II, thus providing evidence that the wires were properly placed.

It is helpful for some purposes to think of the leads of Einthoven's triangle as meeting at a central point. In **Figures 7.3** and **7.4**, leads I, II, and III are shown pushed inward, resulting in what is sometimes called a *triaxial diagram*. The utility of this will become apparent later. For now it is important to begin thinking of these leads as they are shown in Figure 7.4.

Although much information is obtained from the original three leads (I, II, and III), it became clear over time that additional electrical views (i.e., leads) would be helpful. The next three leads invented were aVR, aVL, and aVF. Leads other than I, II, and III are technically not simple bipolar leads. For example leads aVR, aVL, and aVF have their voltage increased to increase clarity, and neither these leads nor the chest leads have a single electrode functioning as a negative pole. Because of this they are sometimes referred to as unipolar leads. A technical discussion of the nature of these leads is beyond the scope of this book. Fortunately, detailed knowledge of the technical aspects of these issues is not needed to successfully interpret cardiograms, and for conceptual purposes it is quite useful (although technically incorrect) to think of all 12 leads as simple bipolar leads.

Figure 7.5 shows all six limb leads arranged to meet at a central point in what is often called a *hexaxial diagram*. The name of the lead (e.g., I) is placed at the positive pole and the negative pole of each lead is labeled with a minus sign. The six limb leads (I, II, III, aVR, aVL, aVF) are all obtained using only four

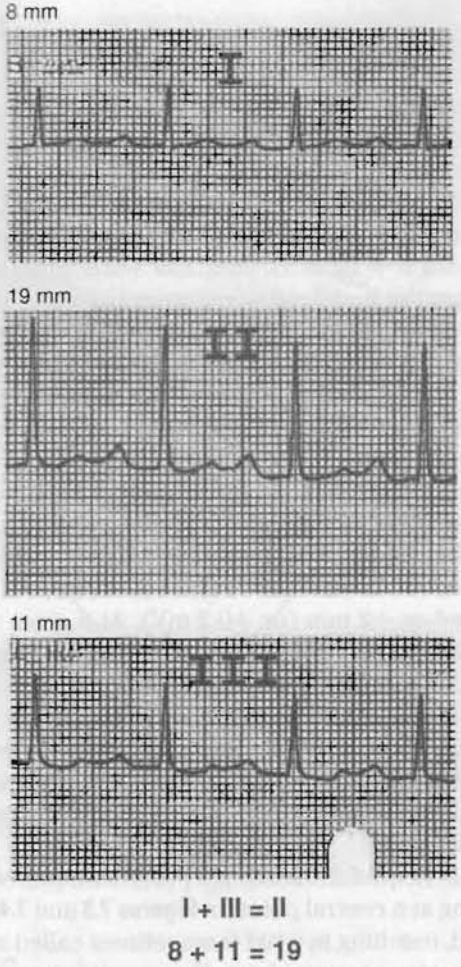

8 mm
I
19 mm
II
11 mm
III
I + III = II
8 + 11 = 19

FIGURE 7.2 I + III = II.

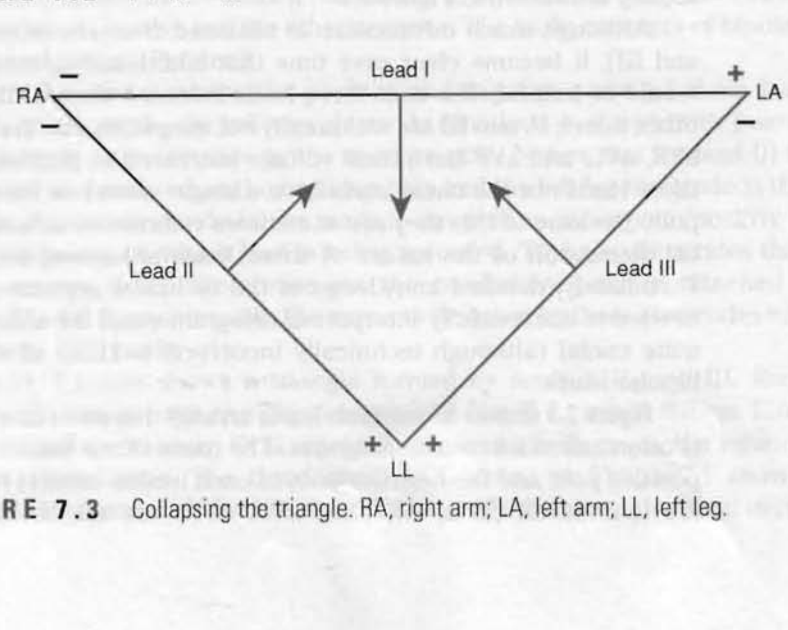

Lead I
RA
LA
Lead II
Lead III
LL
−
−
+
−
+
+

FIGURE 7.3 Collapsing the triangle. RA, right arm; LA, left arm; LL, left leg.

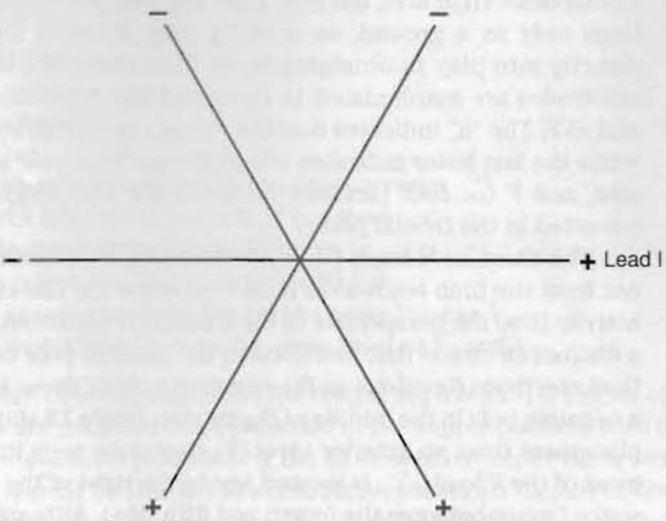

–
–
–
+ Lead I
+
Lead III
+
Lead II

FIGURE 7.4 Triaxial diagram.

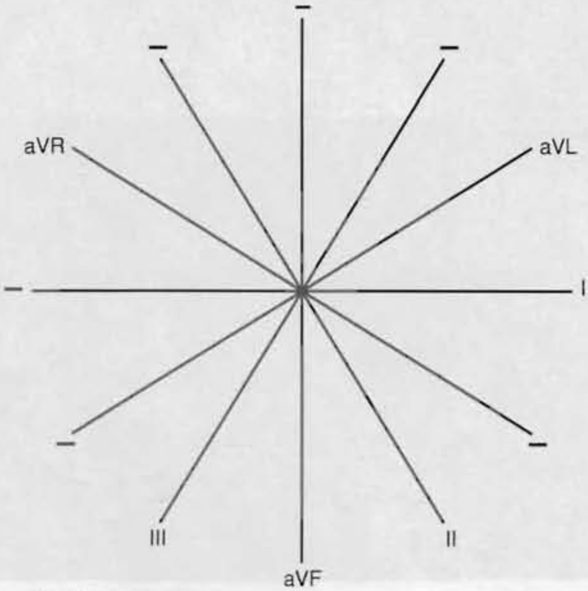

aVR
aVL
I
III
II
aVF

FIGURE 7.5 Hexaxial diagram.

electrodes—right arm, left arm, right leg, and left leg. The right leg electrode functions only as a ground, so actually only three of these four electrodes come directly into play in obtaining input from these six leads. Inputs from the limb electrodes are manipulated to construct the three augmented leads—aVR, aVL, and aVF. The "a" indicates that the voltage recorded by these leads is augmented, while the last letter indicates where the positive pole is: R for right arm, L for left arm, and F for foot (actually between the two feet). All of the limb leads are recorded in the frontal plane.

The chest, or V, leads (V_1–V_6) entered the ECG realm last of all. They are different from the limb leads in at least two respects. The chest leads record electrical activity from the perspective of the transverse (horizontal) plane, and each lead has a distinct electrode that functions as the positive pole of that lead. Although no distinct electrode functions as the negative pole of these leads, it is useful to imagine a negative pole in the middle of the thorax. **Figure 7.6** shows the chest lead electrode placement from an anterior view (V_6 cannot be seen in this figure). The rightward most of the V leads, V_1, is located just to the right of the sternum in the fourth interspace (space between the fourth and fifth ribs). Although the most rightward, obviously this lead is actually close to the centerline of the chest (midsagittal line). The standard ECG is mainly focused on the left heart, so relative to the left heart this lead is "rightward." The V leads then proceed to the left with the most leftward being V_6, which is in line with the middle of the axilla on the left side. For a general orientation, it is useful to conceive of V_1 and V_2 as close to the intraventricular septum, V_3 and V_4 as "anterior" in relation to the left ventricle, and V_5 and V_6 as "lateral" in relation to the left ventricle. The specific placement of all of the electrodes for a 12-lead ECG is described below.

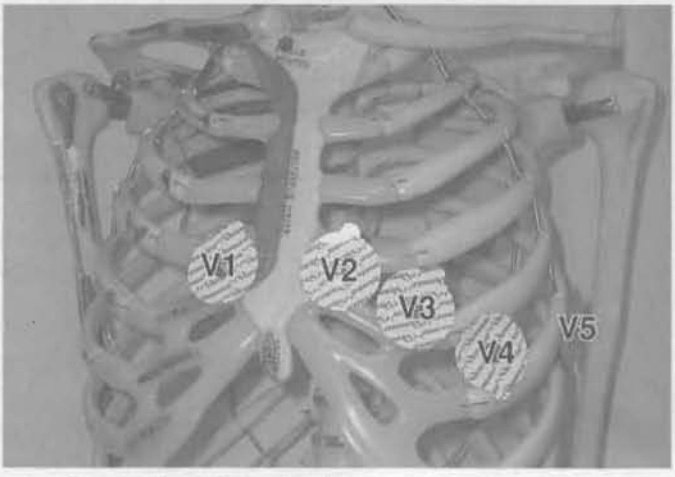


FIGURE 7.6 Anterior view of chest leads.

Electrodes

RA	right arm
LA	left arm
RL	right leg
LL	left leg
V_1	fourth interspace just right of the sternum
V_2	fourth interspace just left of the sternum
V_3	midway between V_2 and V_4
V_4	fifth interspace left midclavicular line
V_5	left anterior axillary line on the same level as V_4
V_6	left midaxillary line on the same level as V_4 and V_5

The placement described above is for resting supine ECGs. Unless otherwise indicated, ECGs are assumed to be recorded in the supine position with the electrodes placed in the standard positions. If the ECG is taken under other conditions (e.g., standing) it should be labeled as such. Some situations dictate different electrode placement. For example, when the ECG is recorded during an exercise stress test the limb leads must be moved as they present a tripping hazard and the tracing would be unreadable due to artifact. In such cases the chest electrodes are placed in the normal positions, but the limb electrodes are moved onto the torso, as described below and shown in **Figure 7.7.** If a nonstandard placement such as the

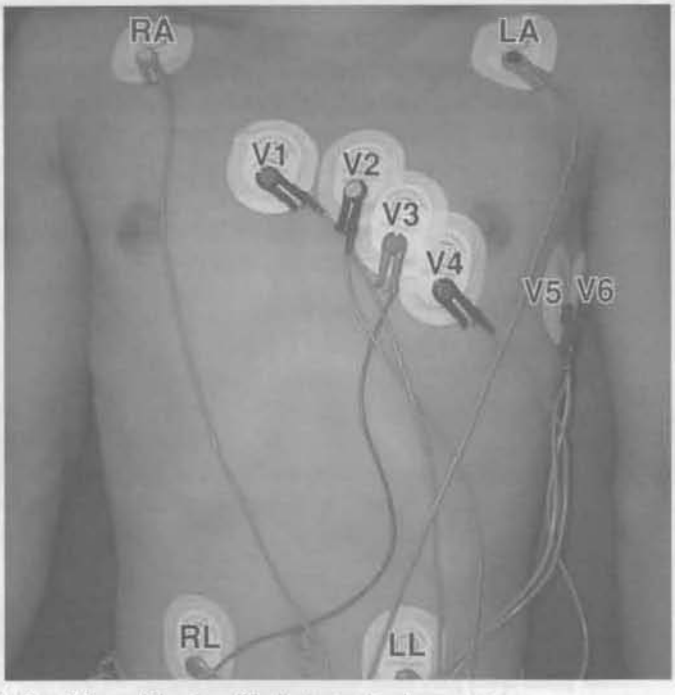


FIGURE 7.7 Mason-Likar (modified) electrode placement.

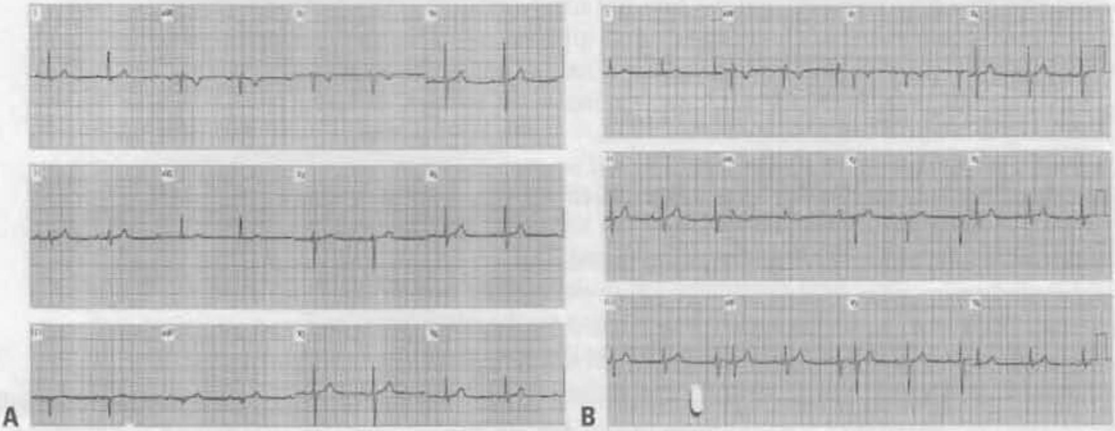


FIGURE 7.8 ECG changes from modified lead placement. **A:** Standard. **B:** Mason-Likar.

Mason-Likar is used it should also be noted on the cardiogram. Thus, an ECG recorded with the modified placement and patient standing might be marked "standing Mason-Likar." Often moving the limb electrodes to the modified placement has little effect on the cardiogram. However, as illustrated in **Figure 7.8,** significant changes may occur. It may therefore be best to record an ECG with standard electrode placement and the patient supine for comparison if nonstandard postures and electrode placements will subsequently be done. A comparison of these ECGs can assist in determining if changes on the cardiogram reflect important physiological phenomena or are simply artifacts from nonstandard conditions.

Mason-Likar Lead Placement

The placement for leads in a Mason-Likar placement, also known as the modified or exercise lead placement, are listed here:

LA	left subclavicular fossa
RA	right subclavicular fossa
RL*	right anterior abdominal wall at the point where an imaginary line coming laterally from the umbilicus would intersect with an imaginary line coming inferiorly from the middle of the right clavicle
LL*	left anterior abdominal wall at the point where an imaginary line coming laterally from the umbilicus would intersect with an imaginary line coming inferiorly from the middle of the left clavicle

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*Other placements have been described for RL and LL. We describe one that is acceptable and easy to objectively determine.

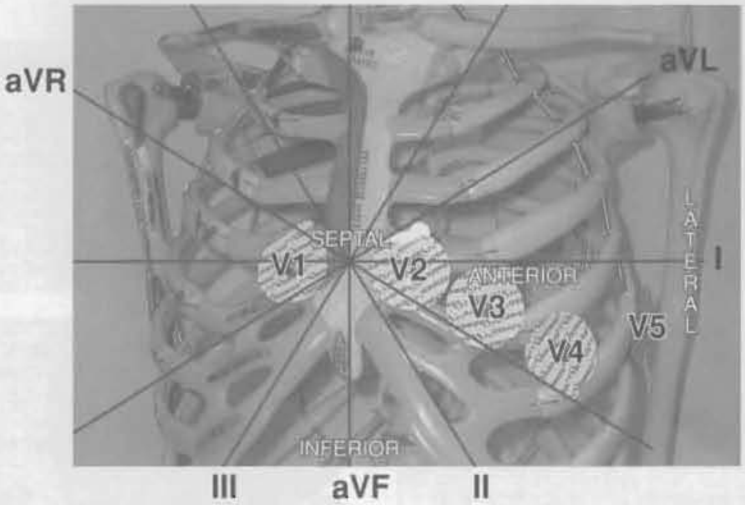


FIGURE 7.9 Leads.

Localization of Leads

Cerain leads typically offer better "views" of certain areas of the heart (**Figs. 7.9** and **7.10**). As the positive poles of leads II, III, and aVF all point downward (straight down in the case of aVF, and obliquely downward in the cases of II and III), these leads together are known as the *inferior leads.* Leads I and aVL are oriented (left) laterally so they are included with V_5 and V_6 (V_6 cannot be seen in Fig. 7.9) as lateral leads. Leads V_1 and V_2 are close to the intraventricular septum and V_4 and V_5 are near the anterior surface of the left ventricle. Lead aVR has a unique perspective and therefore does not belong to a "group" as the other leads do **(Table 7.1)**.

TABLE 7.1

Areas of Lead Placement

Area	Leads
Inferior	II, III, aVF
Lateral	I, aVL, V_5, V_6
Septal	V_1, V_2
Anterior*	V_3, V_4

*Some classifications consider V_1, V_2, V_3, and V_4 to be the "anterior" leads.

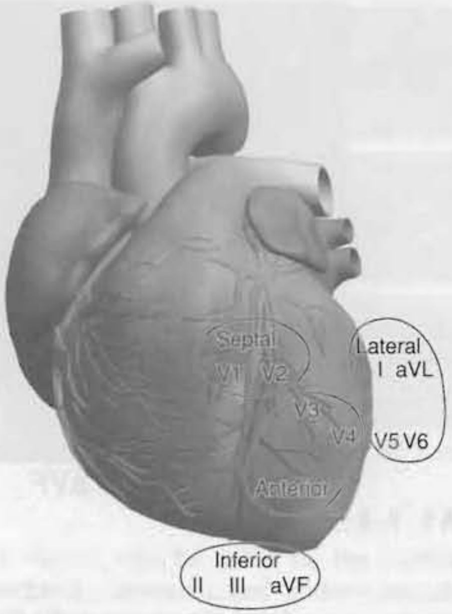


FIGURE 7.10 Approximate areas of the heart associated with different leads.

Normal Patterns

The patterns that appear in the various leads will vary depending on numerous factors such as the mean electrical axis of the heart (Chapter 8), so it is difficult to fully describe what is "normal" in each of the leads. Some general principles can be described. As discussed in Chapter 2, the P waves should be upright (positive) in lead II and negative in lead aVR if normal sinus rhythm is present. In fact, if sinus rhythm is present the P waves should be upright in leads I, II, V_5, and V_6. If the P waves are not positive in all of these leads the term "unusual P wave axis" is sometimes used, this implies that the rhythm may not be sinus in origin and likely is an ectopic atrial rhythm.

Some general concepts concerning the morphology of the QRS complexes in the chest leads are very useful in interpreting cardiograms. Normally the first portion of the ventricles to depolarize is the intraventricular septum, which in most people is activated by a portion of the left bundle branch. Normally the septum depolarizes from left to right, in other words toward the positive poles of V_1 and V_2 (actually this is at least partially due to the anterior vector of depolarization) and away from the positive poles of V_5 and V_6. Therefore, a small r wave of short duration is seen in the right chest leads (V_1 and V_2) and a small q wave of short duration in the left chest leads (V_5 and V_6). Because this is caused by septal activation, the small r in the right chest leads is often referred to as a *septal r wave* and the small q in the left chest leads as a *septal q wave*. After septal activation, the bulk of the right and left ventricles depolarize more or less simultaneously. Since the left ventricle has so much

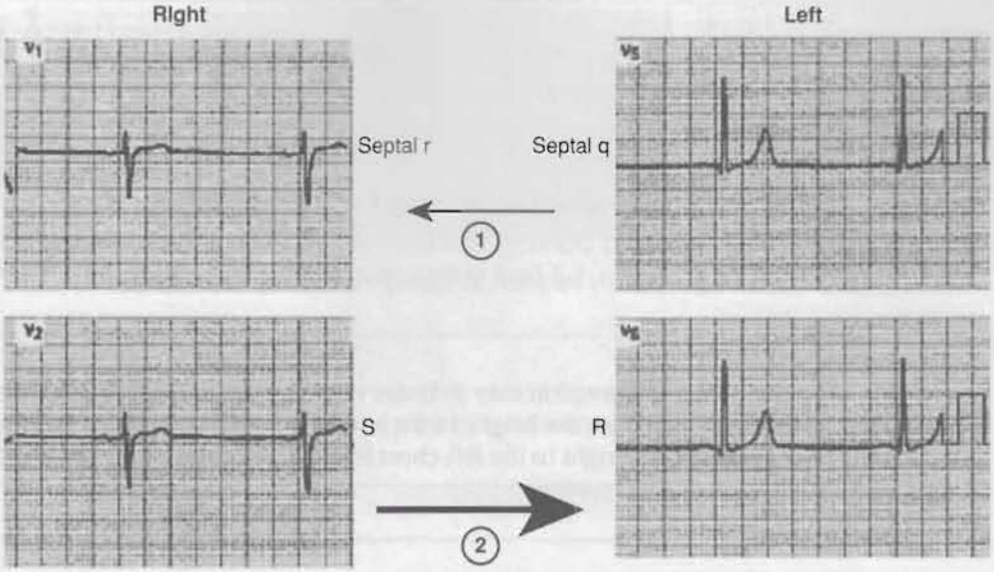


FIGURE 7.11 Normal patterns in the right and left chest leads. The ventricular septum depolarizes first **(1)**, followed by depolarization of the rest of the ventricles **(2)**. *Arrows* indicate the relative size and direction of the net current.

more mass than the right, the mean vector of current is flowing toward the left chest leads and away from the right chest leads. This results in large R waves in V_5 and V_6 and large S waves in V_1 and V_2. **Figure 7.11** illustrates these concepts.

The same electrical event of ventricular depolarization results in different recordings in the various leads because of the position and orientation of the different leads. Leads V_3 and V_4 are in the middle between the right and left chest leads; therefore, the QRS seen in these leads normally has a morphology somewhere in between the rS of V_1 and the qR of V_6. The increase in R wave amplitude from V_1 across to the left chest leads is called *R wave progression*. The size of the R waves in the middle chest leads (V_4 and V_5) is typically intermediate, whereas both of the lateral chest leads (V_5 and V_6) should normally show relatively large R waves (although it is common for V_5 to have a larger R than V_6).

As the pattern shifts from an rS in the right chest leads to a qR in the left chest leads, somewhere, typically in the middle chest leads (V_3 and V_4), the height of the R wave becomes greater than the depth of the S wave. The point (lead) where the R wave becomes as large as or larger than the S wave is called the *transition zone*. Normally, as shown in **Figure 7.12**, transition occurs in V_3 or V_4. If the R wave is as large as or larger than the S wave in V_1 or V_2 this is called *early transition*, if transition does not occur until V_5 or V_6 it is called *late transition*. Ectopic beats are not included in these determinations, so in the late transition example of Figure 7.12 the second QRS complex with the large R wave in lead V_3 would not be counted as it is a premature ventricular complex.

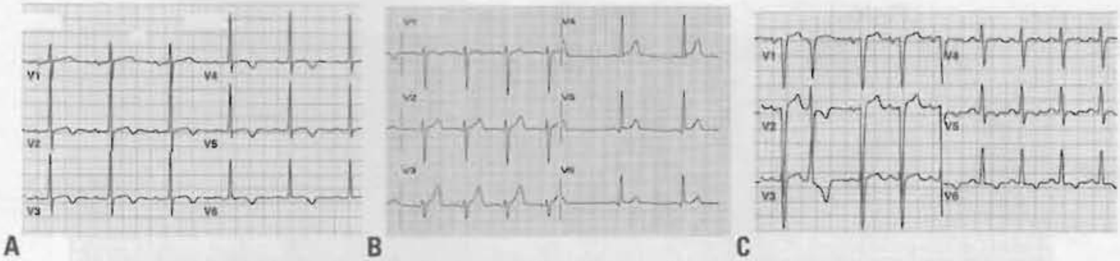
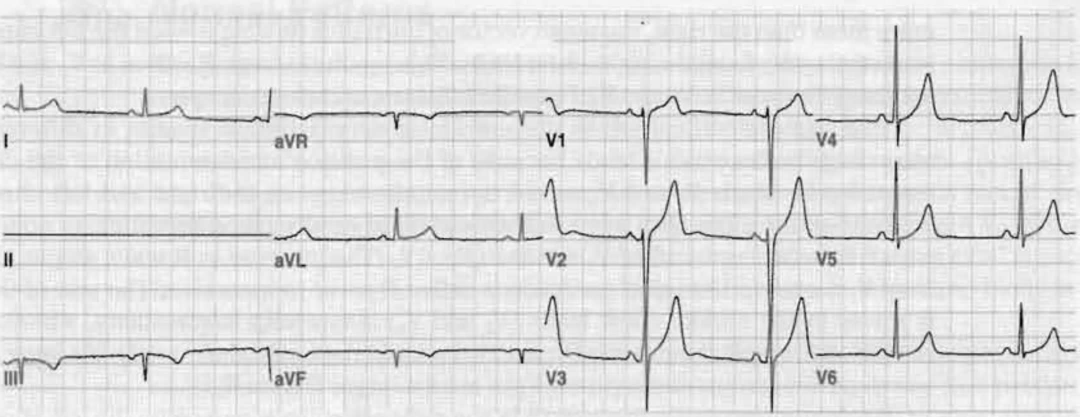


FIGURE 7.12 **A:** Early transition. **B:** Normal transition. **C:** Late transition.

Abnormalities of transition may indicate various conditions to be discussed in later chapters. Sometimes the height of the r waves does not increase significantly moving across from the right to the left chest leads. This is abnormal and is referred to as *poor R wave progression.*

CLINICAL VIGNETTE REVISITED

A student intern under your supervision is puzzled by a resting ECG and wants to know if "the lead II electrode fell off."



Lead II does not have its own separate and distinct electrode. The RA and LL electrodes (along with the RL ground) are used to form lead II, but they are also used for other leads. The I + III = II method indicates no technical error (I is +6 mm, III is –6 mm, and II is 0). Why then is lead II a flat line? (Continued in Chapter 8.)

Quiz 7

1. The six limb leads are obtained using only four electrodes. How is this possible?

2. Why does the depolarization of the ventricles typically result in an rS complex in lead V_1, while the same electrical events are seen as a qR in lead V_6?

3. Which leads are the "limb leads" and which leads are the "chest leads"?

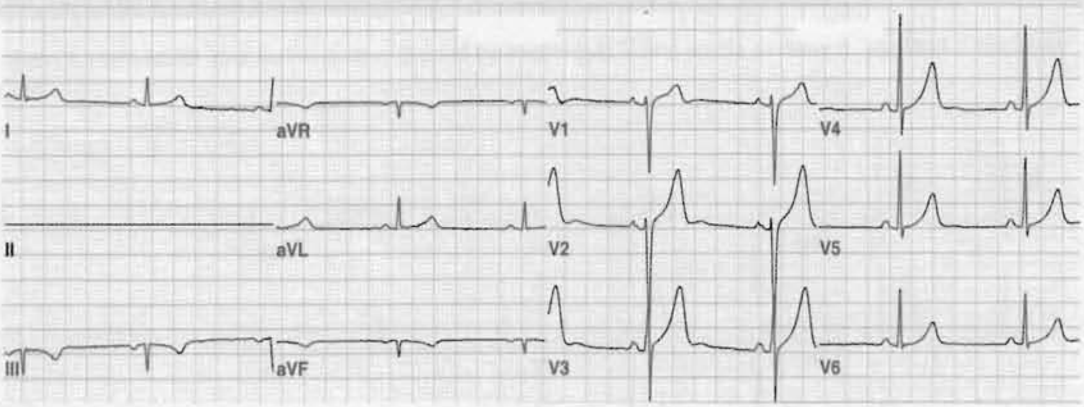
8

AXIS

- Normal Axis
- Axis Quadrant
- Quantitative Description of Axis
- Determining Axis
- Indeterminate Axis

CLINICAL VIGNETTE

If the flat line in lead II is not caused by technical error, what is it a result of?



Axis is a term used to describe the net direction that the current of ventricular depolarization is traveling in the frontal plane. Depolarization of the ventricles is a complex event, occurring in three dimensions with current traveling in numerous directions simultaneously. The net direction of this current (in the frontal plane) can be determined from a standard 12-lead ECG using an extension of some basic principles described previously.

Recall from Chapter 2 that current moving toward the positive pole of a lead will result in a positive deflection; current moving away from the positive pole (toward the negative pole) of a lead is recorded as a negative deflection; and current moving perpendicular to the orientation of a lead causes a deflection with a net voltage of zero (either a flat line or a complex with equal positive and negative components). We can use these principles to determine the net vector of electrical currents recorded on a cardiogram.

Normal Axis

For many purposes it is only of interest whether the axis is normal, deviated to the left or right, or extremely deviant. If the axis is "normal," this can usually be determined very readily by simple inspection of leads I and aVF. If the overall QRS complexes in these leads are positive then the axis is normal. Look at the QRS complexes of leads I and aVF of **Figure 8.1.** They are very positive, consisting mainly of

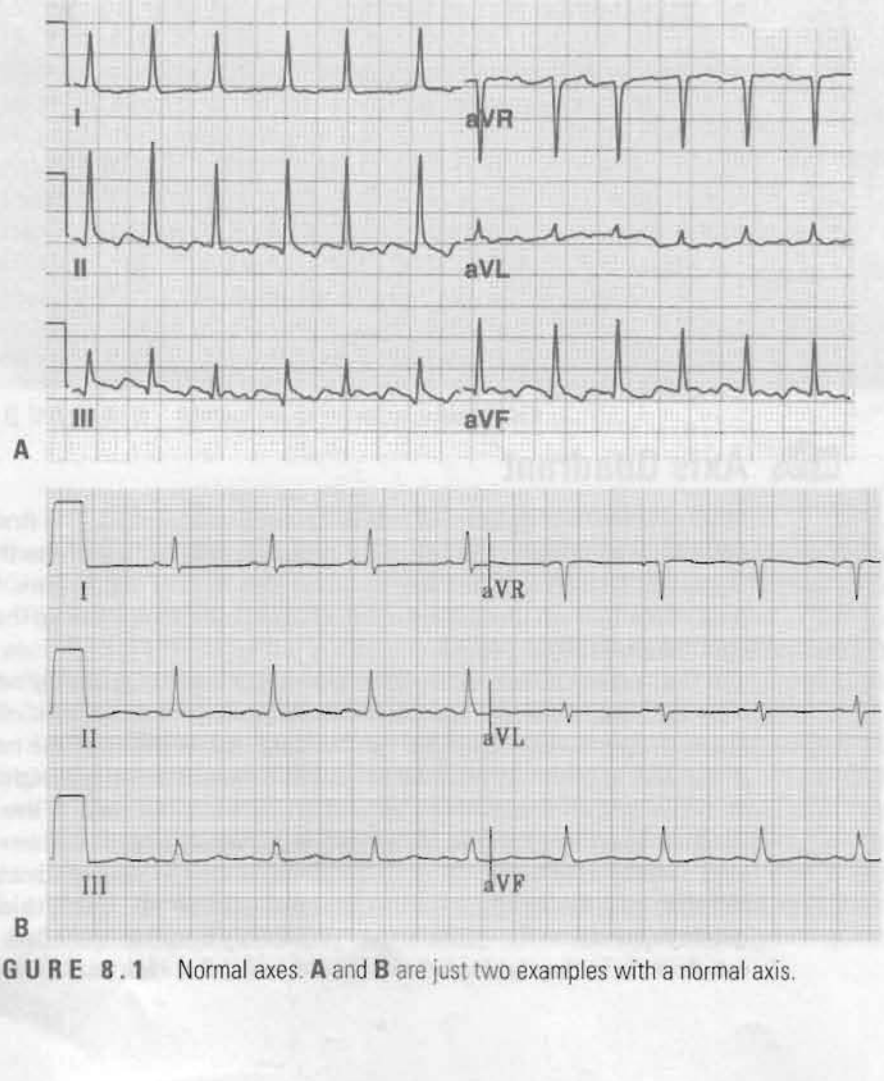


FIGURE 8.1 Normal axes. **A** and **B** are just two examples with a normal axis.

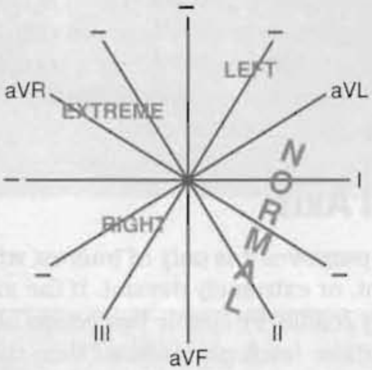


FIGURE 8.2 Axis quadrants.

relatively large R waves. This implies that the net vector of ventricular depolarization is traveling toward the positive poles of these leads. The axis need not be heading directly at the positive pole, and obviously could not simultaneously be heading directly at the positive poles of both of these leads, but rather is oriented more toward their positive poles than away from them. If this were the case for leads I and aVF the net vector (axis) of the current falls within the range considered normal.

Recall that depolarization of the heart normally proceeds down and to the left. This is the basis for the normal axis quadrant **(Fig. 8.2)**. With many cardiograms this simple, cursory inspection of leads I and aVF to establish that the QRS complexes are more positive than negative in these leads is all that is needed to establish that the axis is normal.

Axis Quadrant

More precise descriptions of axis are sometimes needed. The first step is establishing which "quadrant" the axis is in. A simple way to determine this is illustrated in **Figure 8.3.** This technique uses the arms in a kind of semaphore. Since the positive pole of lead I is on the left arm, the left arm is extended out to the left if the QRS in lead I is generally positive.

Conversely, if the overall QRS complex in lead I is generally negative, extend the right arm out to the right (away from the positive pole). Based on the hexaxial diagram, the positive pole of aVF can be imagined as between the two feet. Therefore, if the QRS is generally positive in lead aVF extend an arm straight down; if the QRS in aVF is negative extend the arm up. In between the arms is the quadrant wherein the axis lies. For example, if the QRS is generally negative in lead I and positive in lead aVF the axis lies in the right quadrant. If the QRS is negative in both leads I and aVF the axis lies in the extreme quadrant and so on. Using this method, one can quickly determine the quadrant. As previously noted this is often all that is of interest. An axis in the right quadrant is said to exhibit *right axis deviation* (RAD), one

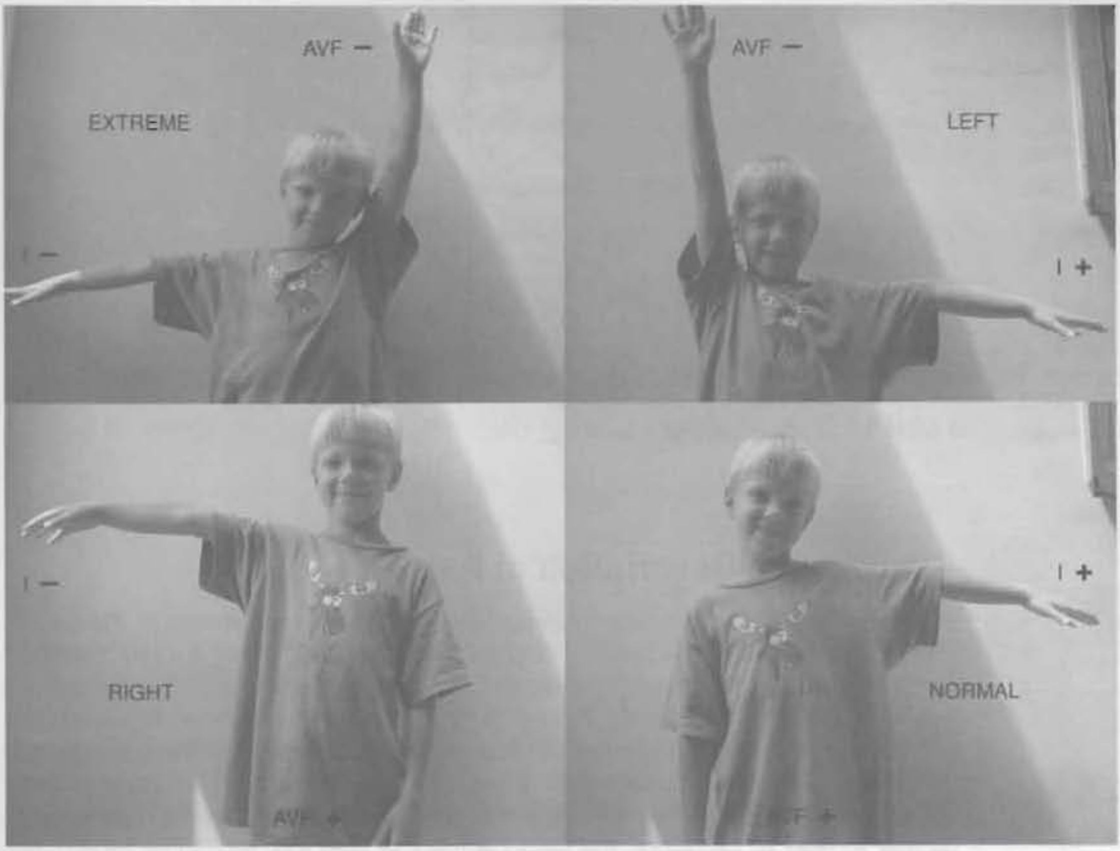


FIGURE 8.3 Method for determining axis quadrant.

in the left quadrant *left axis deviation* (LAD), one in the extreme *quadrant extreme axis deviation*, and an axis in the normal quadrant is said to have a *normal axis*.

Figure 8.4 is the hexaxial diagram previously described in Chapter 7. The name of each lead is placed by the positive pole of the lead. An additional feature is that the circle described by this diagram has been subdivided into four parts: normal, left (axis deviation), right (axis deviation), and extreme (axis deviation). Notice that the four axis quadrants are not equal; the normal quadrant is expanded. Many apparently normal individuals have an axis that falls in the left quadrant slightly above the positive pole of lead I, so it is common to expand the normal axis zone as shown. This complicates matters slightly as it is possible for an axis in the left quadrant to be considered normal. Some authorities use zones of equal size, avoiding this problem. In such a system the normal quadrant is not expanded, and any axis in the left quadrant would be considered LAD. Here the expanded normal quadrant is used.

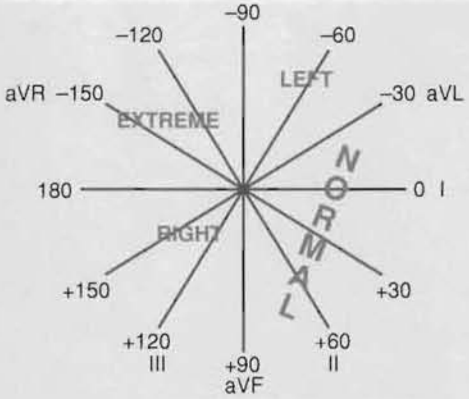


FIGURE 8.4 Axis degrees. Normal −30 to +90, Right +90 to 180, Extreme 180 to −90, Left −30 to −90.

Quantitative Description of Axis

The axis can also be described numerically using a system shown in Figure 8.4. The axis circle is divided into 360 degrees. By convention there are 180 "positive" degrees and 180 "negative" degrees, and the positive pole of lead I is designated as the zero point (0 degrees). Let us assume that the net current of ventricular depolarization in a particular patient is traveling directly toward the positive pole of lead II (+60). This should result in the QRS complex in lead II largely consisting of a positive deflection (i.e., R wave). The orientation of lead aVL is perpendicular to lead II, therefore, the QRS complex in lead aVL should be largely isoelectric. Building on these principles we can determine the axis. First establish the quadrant. The QRS complexes in leads I and aVF of **Figure 8.5** are more positive than negative, therefore, the axis must fall in the normal quadrant. Next determine which of the limb leads (chest leads are not used to determine axis as these leads are not aligned in the frontal plane) shows an isoelectric QRS complex (one that has equal positive and negative components). In Figure 8.5 the positive R wave of aVL is almost identical in height to the depth of the negative S wave, thus the net QRS is essentially isoelectric. Using our basic principles, if lead aVL is isoelectric, then the axis should be perpendicular to it. The axis could be +60 or −120; we have already determined that the axis is in the normal quadrant, so it must be +60.

Examine **Figure 8.6**. The QRS complex in lead I is largely positive, while that in lead aVF is mostly negative. Therefore, the axis must fall in the left quadrant. Notice that none of the limb leads is truly isoelectric (positive and negative components of the QRS complex canceling out to zero). This is often the case and necessitates selection of the lead that is closest to isoelectric. Although the positive and negative components of the QRS complex in lead aVR are not identical, the QRS in this lead is closer to isoelectric than the other limb leads. This means

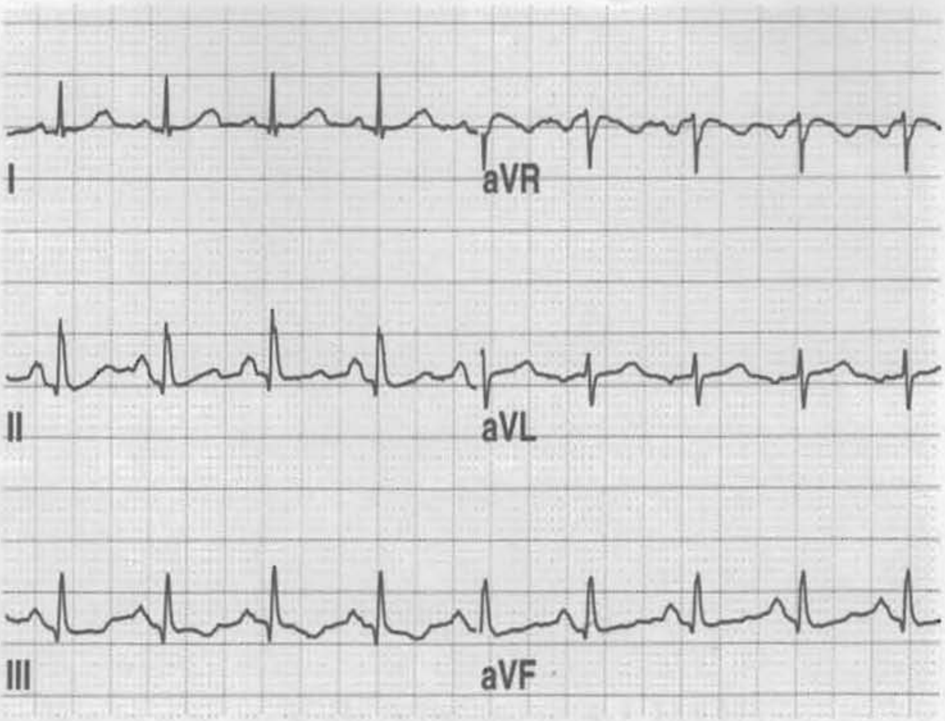


FIGURE 8.5 Normal axis +60.

that the axis is close, but not exactly perpendicular, to this lead (either –60 or +120 degrees). Since we have already established that the axis is in the left quadrant, the axis must be closest to –60 degrees. As the QRS complex in lead aVR is not exactly isoelectric we know that the axis is not exactly perpendicular to this lead; however, the exact axis should be within 15 degrees of the axis obtained using this method (which is perfectly reasonable for most clinical purposes). We could therefore describe the axis in Figure 8.6 qualitatively as LAD and quantitatively as –60 degrees.

If we use the expanded normal quadrant it is possible for the axis to be in the left quadrant yet be considered normal. In **Figure 8.7** the QRS complex is mostly positive in lead I and negative in lead aVF; therefore, the axis is in the left quadrant. Of the limb leads, lead II is the closest to isoelectric. If the axis is in the left quadrant and roughly perpendicular to lead II it must be close to –30 degrees. We would therefore describe this axis quantitatively as –30. If the QRS complex in lead II were perfectly isoelectric, we would know that the axis was exactly –30 degrees and therefore normal. However, since lead II is not exactly isoelectric, the axis is close to but not exactly –30 degrees. If the axis were slightly more

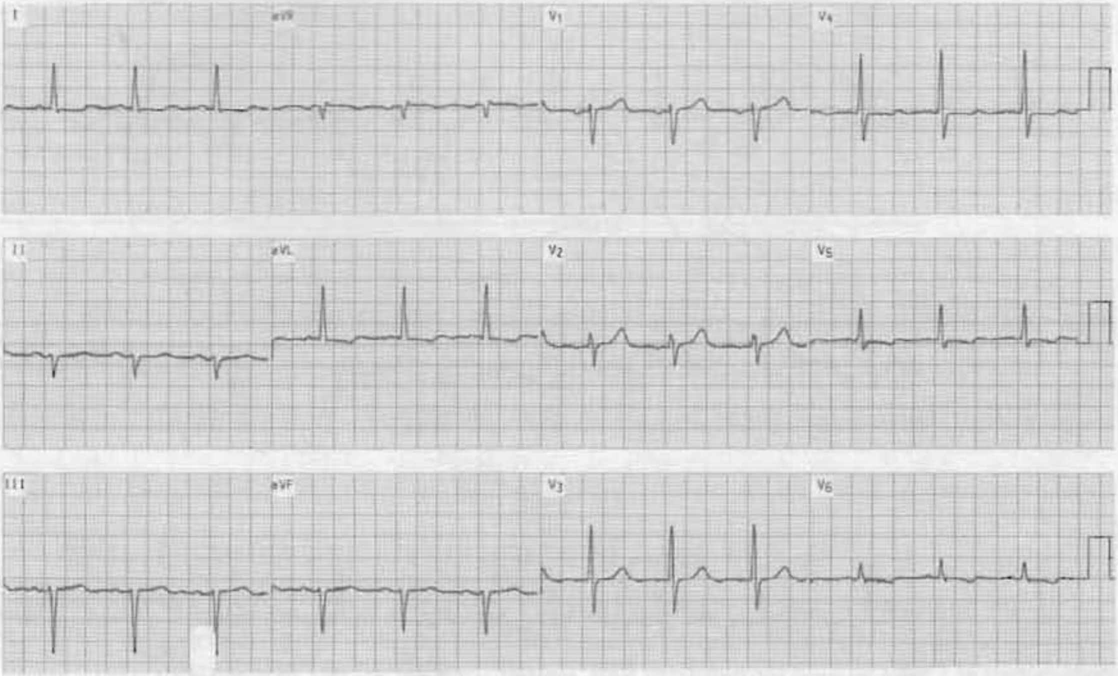


FIGURE 8.6 Left axis deviation −60.

negative than –30 then it would be considered LAD, while if it is slightly more positive than –30 it would be a normal axis. A little reasoning can resolve this dilemma. If the QRS complex in lead II were more positive than negative, which it is in this case, then the axis must be pointing a little more toward the positive pole of lead II (+60) and therefore would fall into the normal quadrant. If the QRS complex in lead II were more negative than positive, then the axis must be directed a little more toward the negative pole of lead II (–120) and therefore LAD would be present.

Figure 8.8 shows an example where the axis is roughly perpendicular to lead aVF (aVF has the most isoelectric QRS complex). Since aVF is used with lead I in order to determine which quadrant the axis is in, this might seem to be a problem. Actually in this case the axis is quite easy to estimate as it must be roughly perpendicular to lead aVF and oriented toward lead I (as the QRS in lead I is clearly more positive than negative), and therefore must be approximately 0 degrees. Since 0 degrees falls within the normal quadrant, the axis is normal. Similarly, if lead I had the most isoelectric QRS complex, then the axis must be roughly perpendicular to lead I. In such cases, if the QRS in lead aVF were positive the axis would be +90, and if the QRS in aVF were negative the axis would be –90.

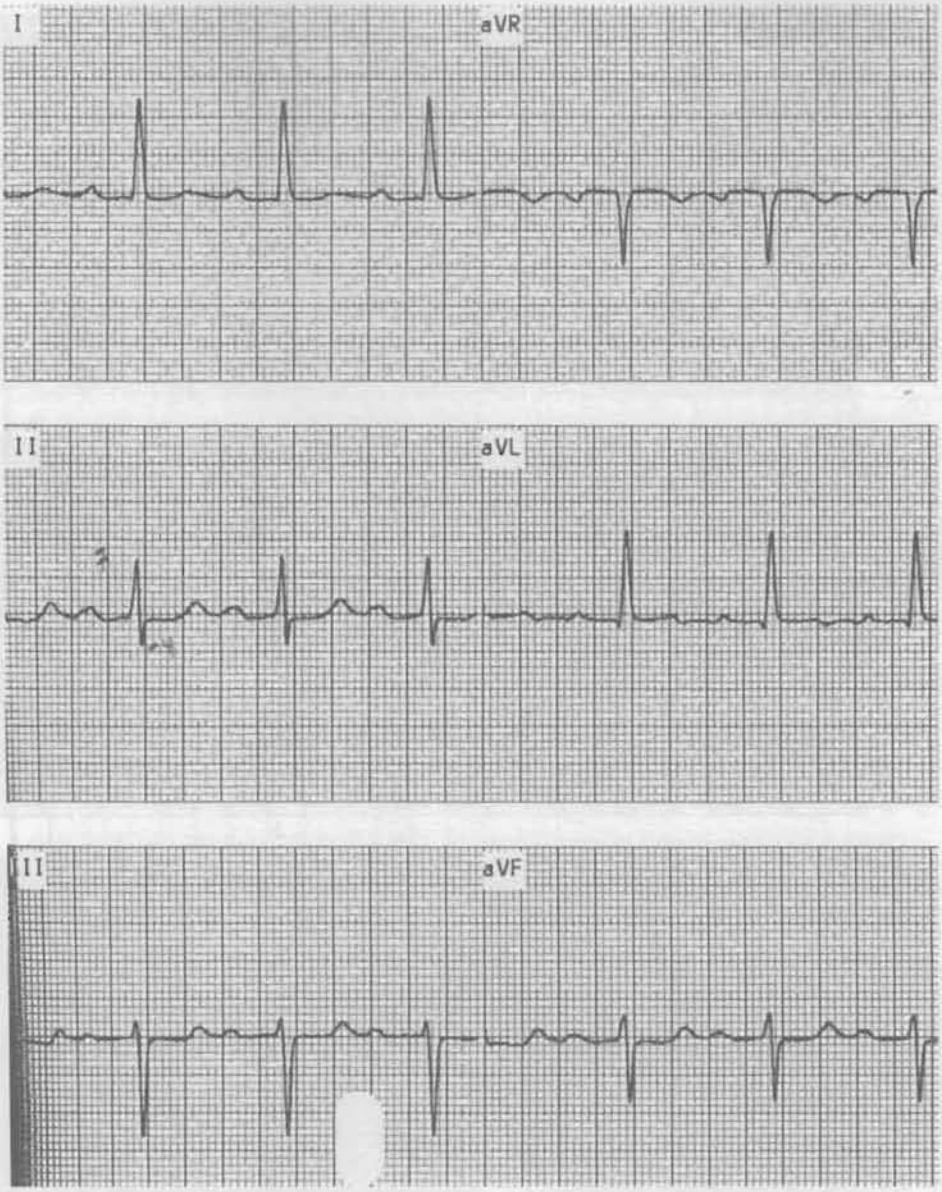


FIGURE 8.7 Left quadrant, normal axis.

II
+7
-4 = +3

aVF
+3.5
-8.0 = -4.5

Figure 8.9 is a cardiogram from a patient in atrial fibrillation. The net QRS complexes in lead I are negative and those in aVF are positive, thus the axis lies in the RAD quadrant. Clearly, lead aVR is the most isoelectric because it has essentially no QRS complexes and the axis lies directly perpendicular to the orientation to this lead. Since we have already established that the axis is in the right quadrant, numerically the axis must be +120.

I +10 −0 = +10

AVF +1 −1 = 0 → zero

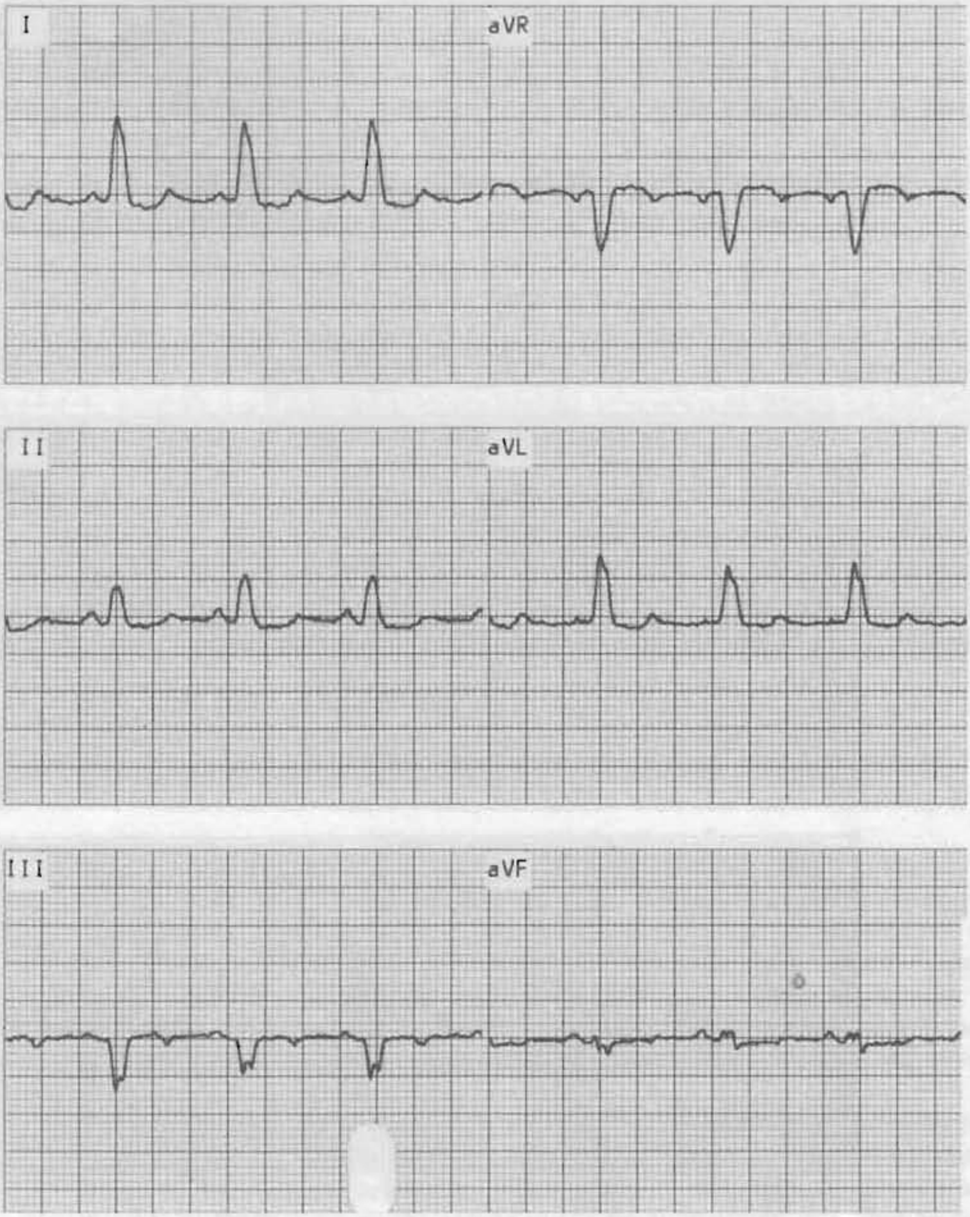

I
aVR
II
aVL
III
aVF

FIGURE 8.8 Axis 0.

+90 to +180

→+ Right Quad

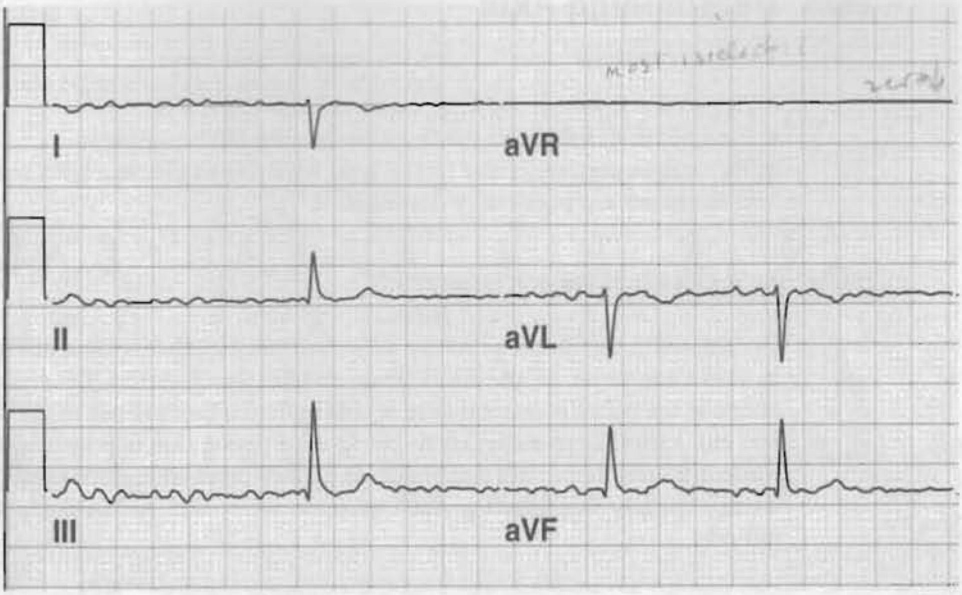

most isoelectric!
zero
I
aVR
II
aVL
III
aVF

FIGURE 8.9 Right axis deviation +120.

+2 -2

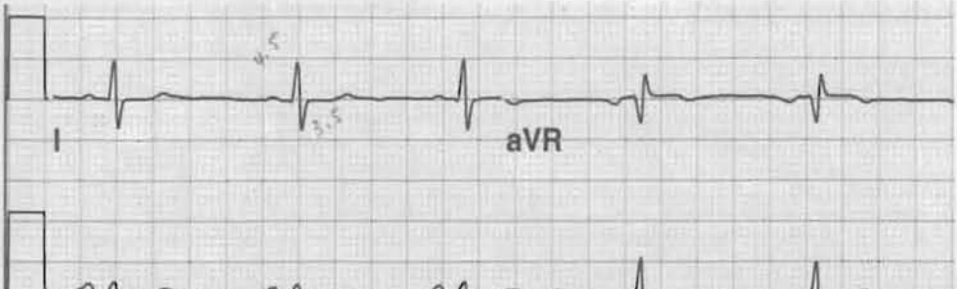

+5
3.5
I
aVR
II
aVL
III
aVF

FIGURE 8.10 Indeterminate axis.

Because all Frontal leads are isoelectric
Heart is not frontal plane
perpendicular to the plane

KSA Determining Axis

The steps to determine the axis are summarized here:

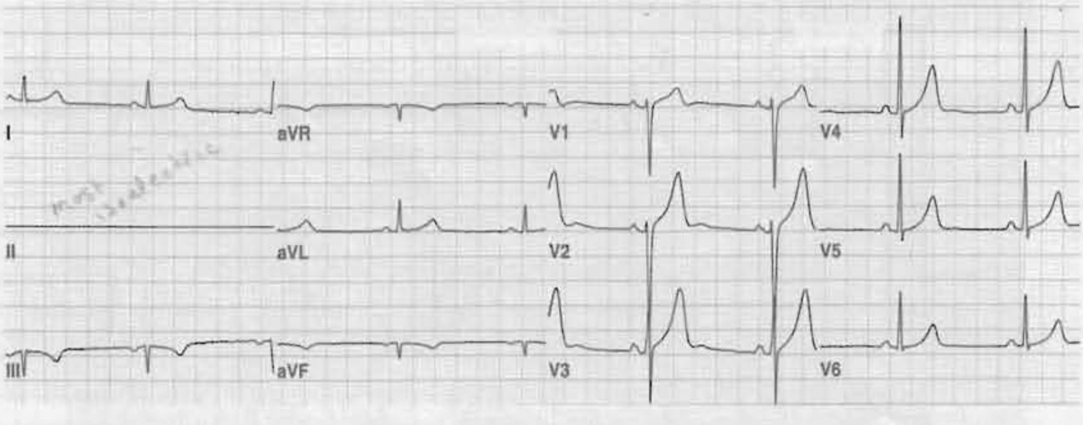
- Determine quadrant by inspection of leads I and aVF.
- Find the limb lead with the most isoelectric QRS complex.
- The axis is perpendicular to the lead with the isoelectric QRS complex and falls in the quadrant previously determined.

KSA Indeterminate Axis

Occasionally, one will encounter a cardiogram like that shown in **Figure 8.10.** Notice in this case most of the limb leads exhibit isoelectric QRS complexes. This is because the mean electrical axis of this patient's heart is not in the frontal plane. In fact the axis is perpendicular to the frontal plane; this is why so many of the limb leads are isoelectric. Normal methods cannot be used in such cases, and the axis is usually simply described as an *indeterminate axis* because it cannot be determined.

CLINICAL VIGNETTE REVISITED

If the flat line in lead II is not caused by technical error, what is it the result of?



The "flat line" in lead II occurs because the mean QRS axis (–30 degrees) is directly perpendicular to the orientation of lead II, resulting in an isoelectric QRS complex.

LEFT Axis -30° to -90°

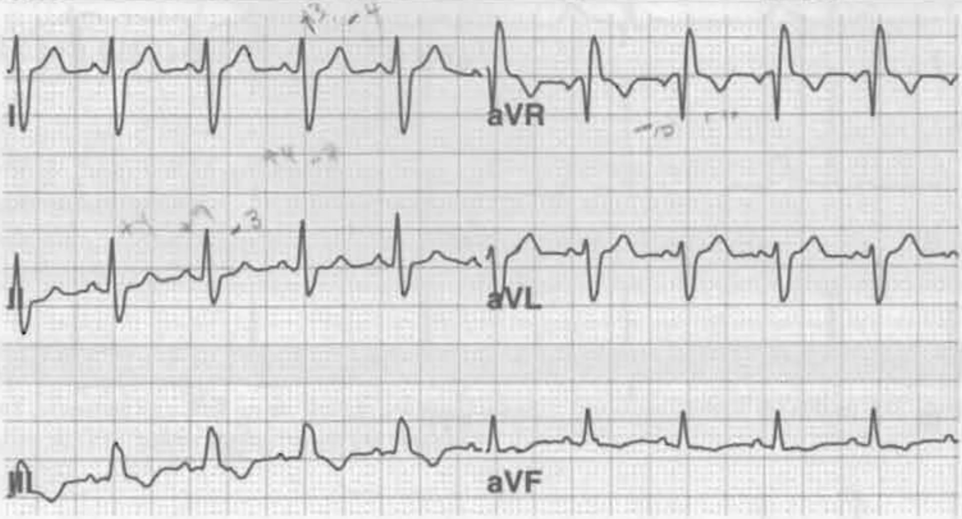
-30

Quiz 8

1. Describe the axis (quantitatively and qualitatively).

QUAD
Right Axis (between +90 and +180)

AVR most Isoelectric; Perpendicular to AVR +120°
III



I
aVR
II
aVL
III
aVF

Right Ax

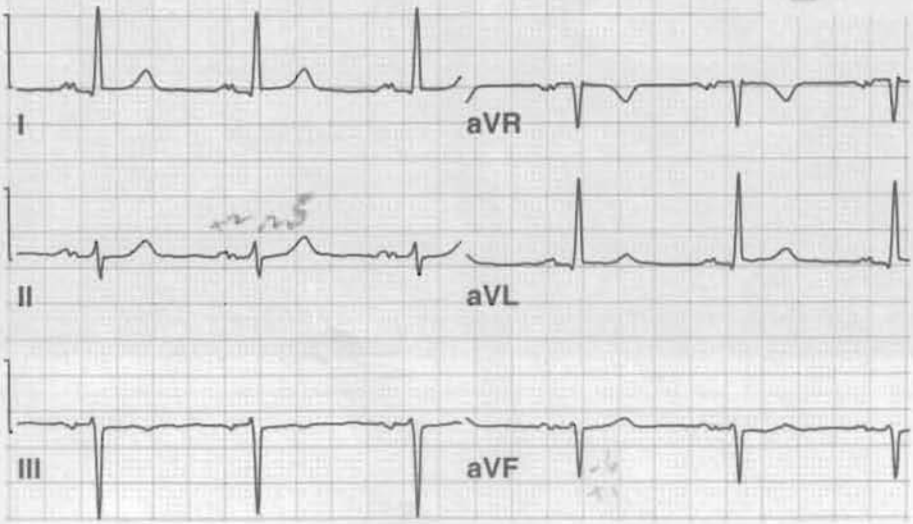
But

+90 and –

2. Describe the axis (quantitatively and qualitatively).

I + QUAD
Left Axis (–30° And –90°)

AVF – AVL = –30° lead II more – Than + Therefore lean toward LAD

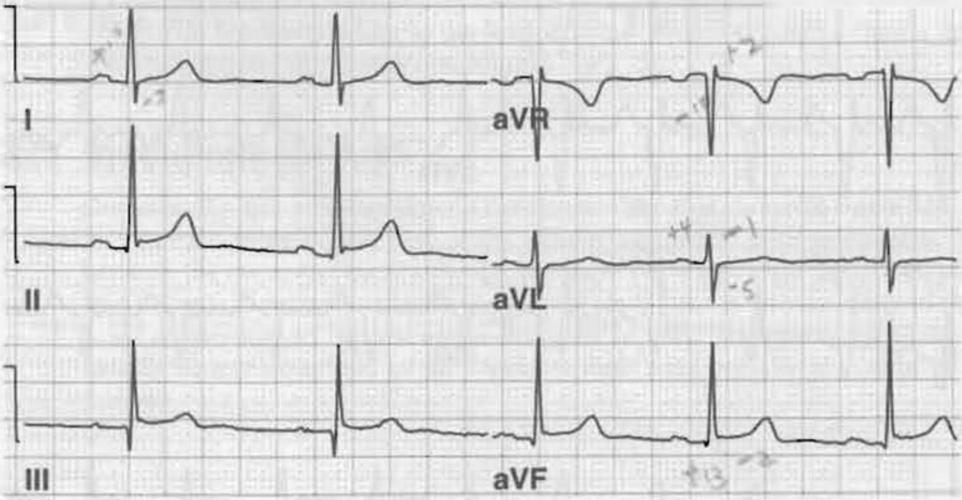


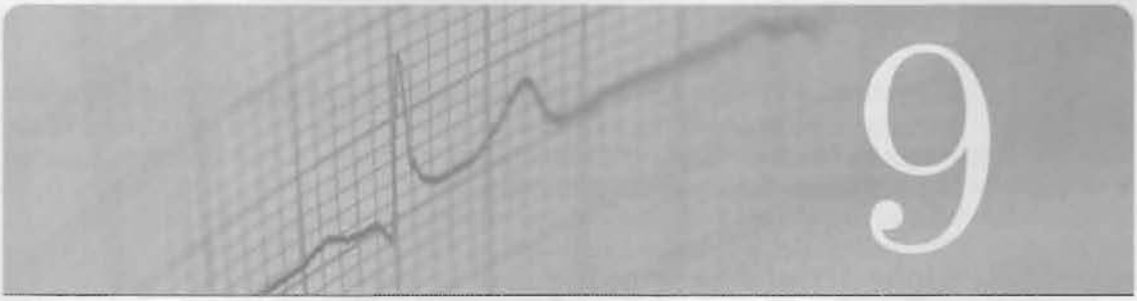
I
aVR
II
aVL
III
aVF

3. Describe the axis (quantitatively and qualitatively).

I = + Normal (Bet +90 And -30)

AvF = + aVL iso = +60


I
II
III
aVR
aVL
aVF
-7
+2
-14
+4
-1
-5
+13
-2



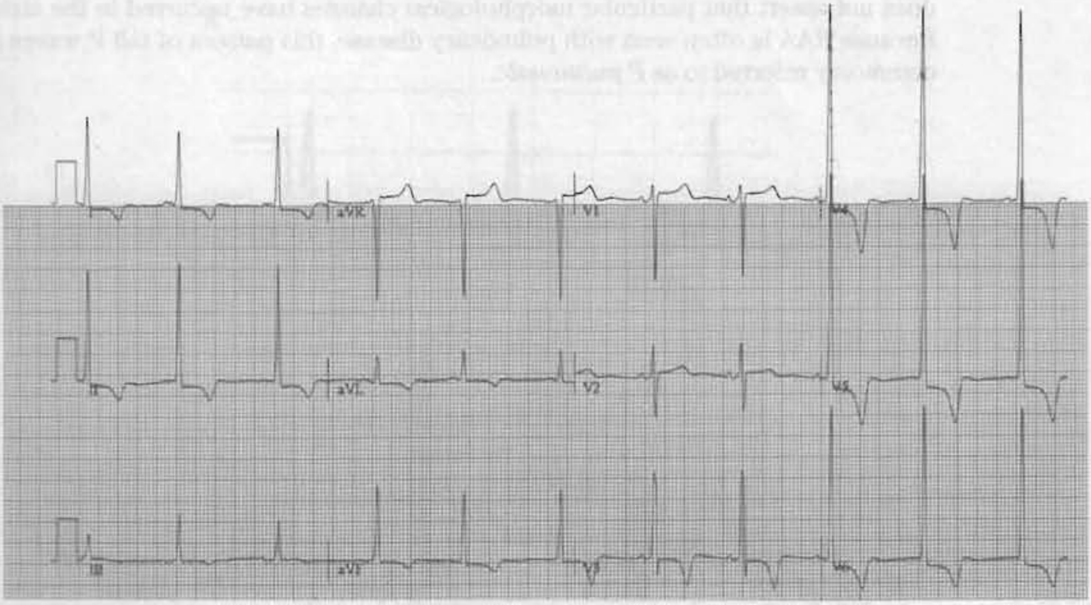
9

HYPERTROPHY

- Right Atrial Abnormality
- Left Atrial Abnormality
- Right Ventricular Hypertrophy
- Left Ventricular Hypertrophy
- Repolarization Abnormalities
- Multichamber Hypertrophy

CLINICAL VIGNETTE

A patient presenting for a plain stress test has inverted T waves in numerous leads on the resting ECG. Should any extra steps be taken before placing him on the treadmill?



The patterns seen in myocardial hypertrophy may be summarized quite simply and logically: hypertrophy of the atria leads to larger P waves, while hypertrophy of the ventricles leads to larger QRS complexes. Of course a few specifics must be learned, but the basic premise is that simple.

KSA Right Atrial Abnormality

Because the sinoatrial node is located in the right atrium, normally the right atrium begins to depolarize slightly before the left atrium. An enlarged right atrium takes longer to depolarize, and therefore a significant portion of the electrical activity of an enlarged right atrium occurs as the left atrium is depolarizing. These electrical signals (right and left atrial) add together, resulting in an abnormally tall, and often pointy ("peaked") P wave **(Fig. 9.1)**. The overall time of atrial depolarization is not changed as the left atrium depolarizes normally and the lengthened depolarization of the right atrium occurs more or less concurrently with the depolarization of the left atrium. Look for these tall and possibly peaked P waves in lead II. A P wave ≥0.25 mV (2.5 mm) is considered abnormally tall.

Sometimes the ECG shows normal P waves when the right atrium is in fact enlarged, and sometimes the P waves are abnormally tall when the atria are of normal size, therefore the term *right atrial abnormality* (RAA) is probably preferable to the term right atrial enlargement as the former indicates an ECG finding, but does not assert that particular morphological changes have occurred in the atria. Because RAA is often seen with pulmonary disease, this pattern of tall P waves is commonly referred to as *P pulmonale.*

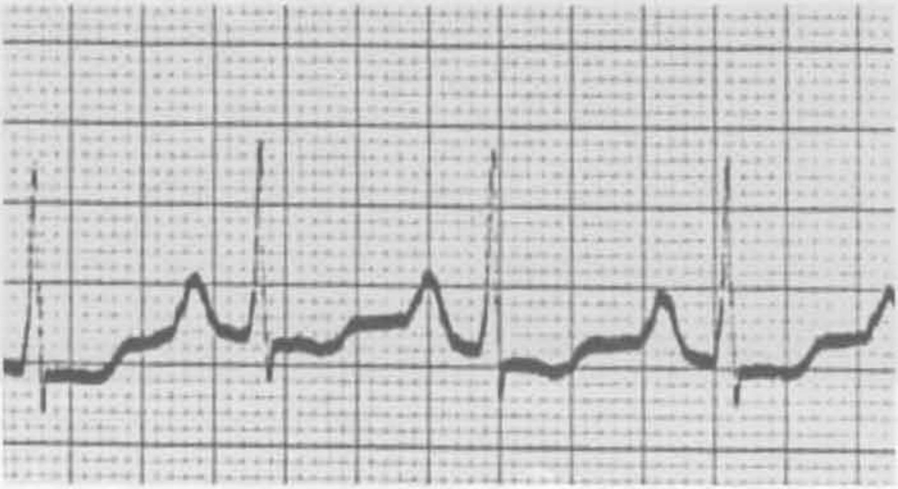


FIGURE 9.1 Right atrial abnormality (enlargement).

Left Atrial Abnormality

When the right atrium is of normal size but the left atrium is enlarged, a wide P wave, often with a notch in the middle, may be seen. The first part of this wide P wave represents the normal electrical activity of the normal right atrium (recall that the right atrium begins to depolarize before the left atrium). The enlarged left atrium then takes longer to depolarize, and therefore the latter part of the P wave lengthens, resulting in the abnormally long P wave **(Fig. 9.2)**. Since most of the electrical activity of the atria is not occurring concurrently the electrical forces do not add together, resulting in a tall P wave as in RAA. Rather most of the additional electrical activity of the left atrium occurs after the right atrium has depolarized, resulting in a long P wave in lead II (≥120 ms). A common cause of *left atrial abnormality* (LAA) is mitral valve disease; therefore, the term *P mitrale* is often used to describe this pattern.

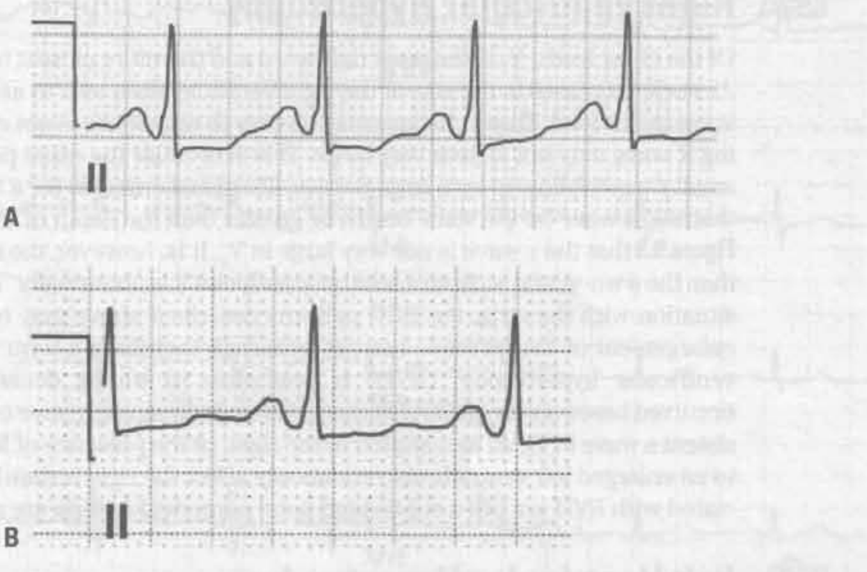


FIGURE 9.2 Left atrial abnormality (enlargement). **A,B:** Wide P waves in lead II. **C:** Wide and deep terminal negative component of P wave in lead V_1.

For LAA leads II and V_1 must be inspected. Normally the P wave in V_1 is biphasic, consisting of an initial positive deflection, representing the depolarization of the right atrium (the vector of which is largely oriented anteriorly and therefore toward the positive pole of V_1) followed by a negative deflection, which represents the depolarization of the left atrium (which is oriented posteriorly and therefore away from the positive pole of V_1). As would be expected, the negative portion of the P wave in V_1 may be altered if the left atrium is enlarged. A "box wide and a box deep" is a common way to refer to the pattern in lead V_1 associated with LAA, as the negative part of the P wave in V_1 must be at least 40 ms (one small box) in width and 0.1 mV (one small box) in depth (Fig. 9.2C).

The appearance of either pattern, a wide P wave in lead II or a "box wide and a box deep" pattern for the negative part of the P wave in V_1, is sufficient to diagnose LAA. The term left atrial enlargement is also used for this condition, but again it is not preferable, because this implies a morphological finding that may not be present.

Right Ventricular Hypertrophy

Of the chest leads, V_1 is the most rightward and therefore closest to the right ventricle. As such, increases in the size of the right ventricle often lead to an abnormally large R wave in this lead. These changes may be easy to miss if one is not careful, as the resulting R wave may not appear very large. This is because the usual pattern in V_1 is a very small r wave followed by a large S wave. The usual criterion for a large R wave in V_1 is that the R wave be the same height or greater than the depth of the S wave. Notice in **Figure 9.3** that the r wave is not very large in V_1. It is, however, the same size or greater than the s wave, and so from a relative standpoint it is abnormally "large." Similar to the situation with the atria, the ECG patterns described above may be seen when actual enlargement of the ventricle has not occurred; therefore, a term like "probable right ventricular hypertrophy" (RVH) is preferable to stating definitely that RVH has occurred based solely on ECG findings. Other findings suggestive of RVH include a persistent s wave in V_6, an axis shifted to the right, or the presence of RAA (factors leading to an enlarged left ventricle also commonly affect the right atrium). The changes associated with RVH are often subtle, and good numerical criteria are not available.

Left Ventricular Hypertrophy

Normally the right chest leads (V_1 and V_2) show a small r wave from septal depolarization followed by a large S wave associated with the simultaneous depolarization of the left and right ventricles, while the left chest leads (V_5 and V_6) show septal depolarization as a small q wave followed by a large R wave associated with depolarization of the ventricles. These patterns occur because the left ventricle has so much more mass than the right ventricle and therefore is electrically predominant, resulting in the net vector of depolarization of the ventricles being directed toward the positive poles of V_5 and V_6 and away from the positive poles of V_1 and V_2. When the left ventricle is enlarged this pattern is exaggerated and the R waves in leads V_5 and V_6 and the S waves in leads V_1 and V_2 become even larger. One commonly accepted criterion for left ventricular hypertrophy (LVH) is to add the height

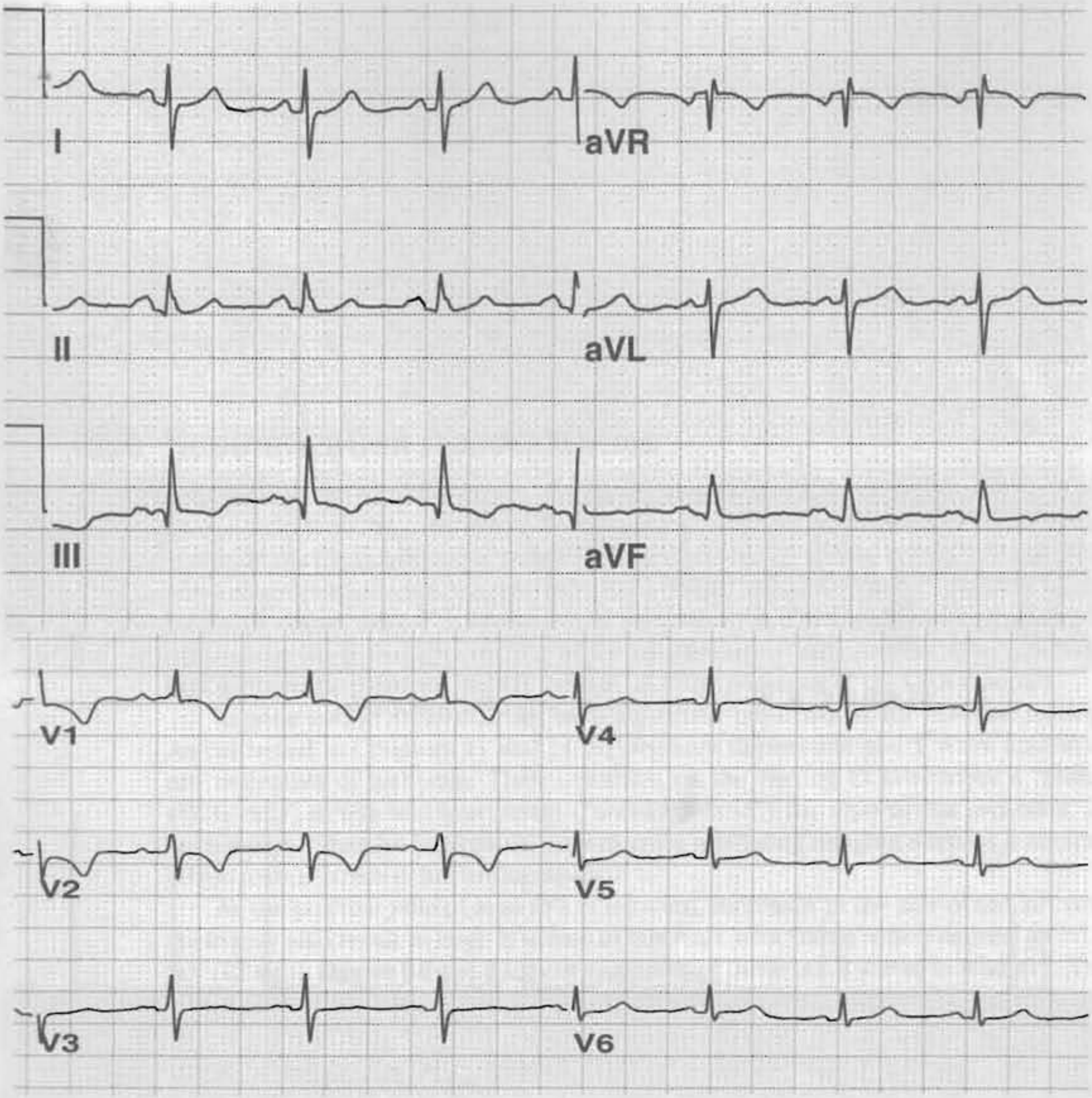


FIGURE 9.3 Right ventricular hypertrophy.

of the tallest R wave in V_5 or V_6 to the depth of the deepest S wave in V_1. If the result is >3.5 mV (35 mm) then "voltage for LVH" is said to be present (**Figs. 9.4** and **9.5**). Usually this criterion is only used for patients 35 years old and older. For patients younger than age 35, a total of 50 or 55 mm is often substituted for 35 mm. Another common formula for LVH voltage is the R in lead I + the S in lead III >25 mm.

LVH is not always pathological. The ECG shown in Figure 9.4 is from a former Olympic rower. In this case the left ventricle likely hypertrophied as a normal response to a long history of intense exercise.

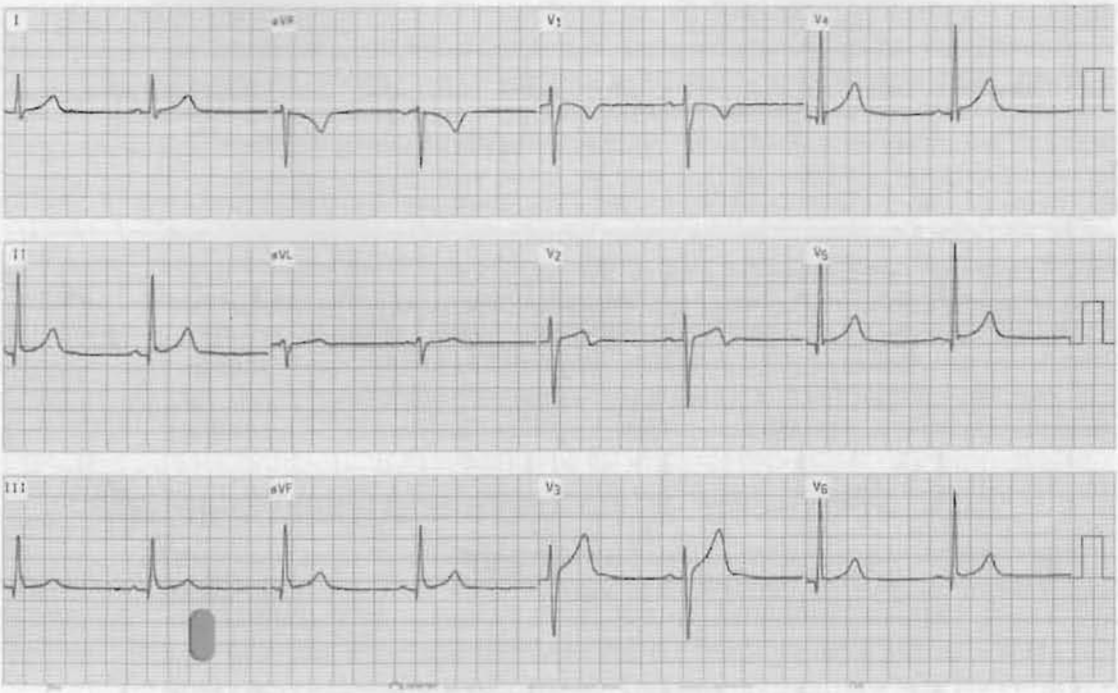

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6

FIGURE 9.4 Voltage for a left ventricular hypertrophy.

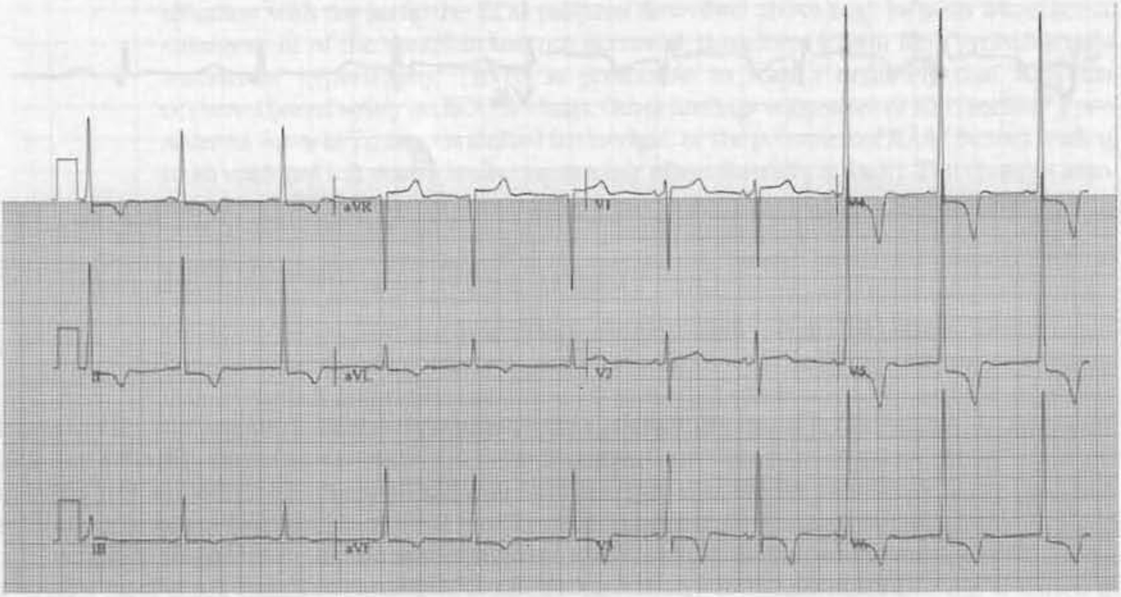

aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3

FIGURE 9.5 Voltage for a left ventricular hypertrophy with "strain."

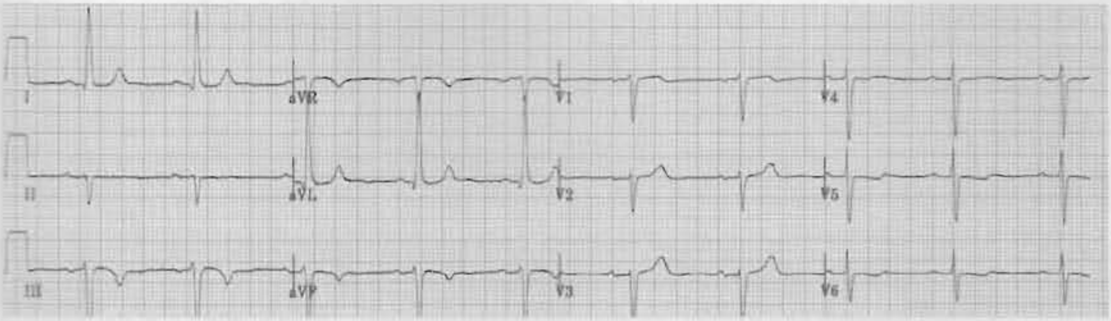


FIGURE 9.6 Voltage for left ventricular hypertrophy in lead aVL.

Repolarization Abnormalities

Enlargement of the left ventricle commonly results in abnormal repolarization. This causes a characteristic pattern of T wave inversion and ST segment depression, which is seen in the leads with tall R waves (V_5 and V_6 in Fig. 9.5, aVL in Fig. 9.7). These ST-T wave abnormalities are often referred to as a "strain" pattern, although the term "repolarization abnormalities" is preferable. If repolarization abnormalities are present it is much more likely that hypertrophy of the ventricle is actually present. Other findings that support the presence of LVH include LAA and widening of the QRS complex.

Repolarization abnormalities have important implications for exercise testing. As discussed in Chapters 11 and 14, ST segment depression and T wave inversion are indicative of ischemia. Their presence on the resting ECG renders a "plain" (ECG only) stress test inaccurate ("nondiagnostic") for myocardial ischemia. In such cases it may be preferable to perform a test using imaging such as a nuclear stress test or stress echocardiography.

As the positive pole of lead aVL is leftward, increases in the size of the left ventricle may also result in large R waves in this lead, with "large" often defined as ≥1.2 mV (12 mm). **Figures 9.6** and **9.7** show examples of these tall R waves in lead aVL. The ECG in Figure 9.7 also shows repolarization abnormalities ("strain") in lead aVL.

Multichamber Hypertrophy

Multiple chambers of the heart may be enlarged. In this case the ECG may show patterns characteristic of more than one type of hypertrophy. For example, in **Figure 9.8A**, the P waves are both wide and tall. Thus, the criteria for both right and left atrial abnormality are present. This pattern is often called *biatrial abnormality*. Figure 9.8B shows wide and tall P waves in lead II (and the terminal negative component of the P wave in V_1 is a "box wide and a box deep"), and height of the R waves in V_5 or V_6 added to the depth of the S waves in V_1 is >35 mm. Therefore, this cardiogram exhibits biatrial abnormality and voltage for LVH.

As previously mentioned, findings on the ECG do not always correlate precisely with the physical size of the heart's chambers. The patient whose ECG is

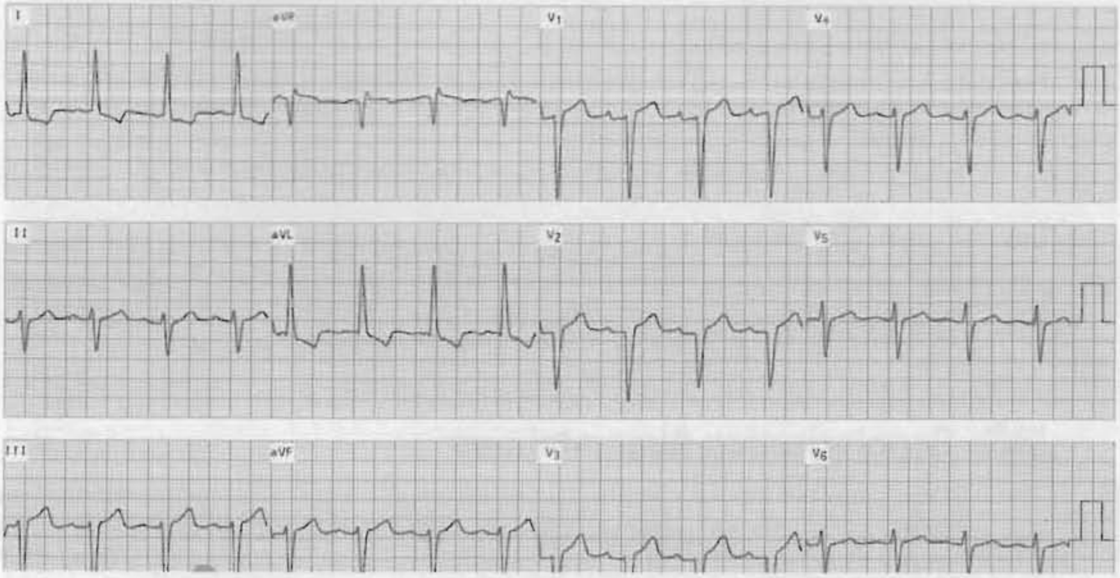

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6

FIGURE 9.7 Voltage for a left ventricular hypertrophy in lead aVL with "strain."

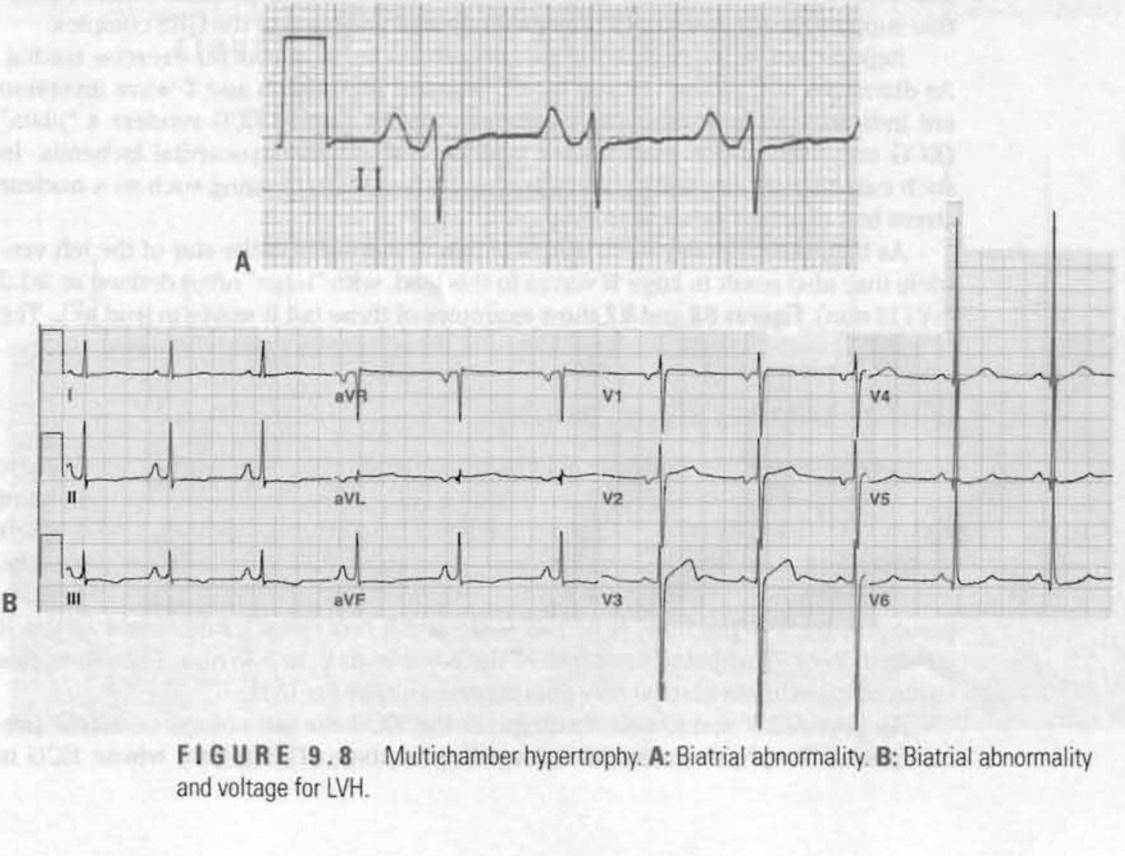

II
A
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
B

FIGURE 9.8 Multichamber hypertrophy. **A:** Biatrial abnormality. **B:** Biatrial abnormality and voltage for LVH.

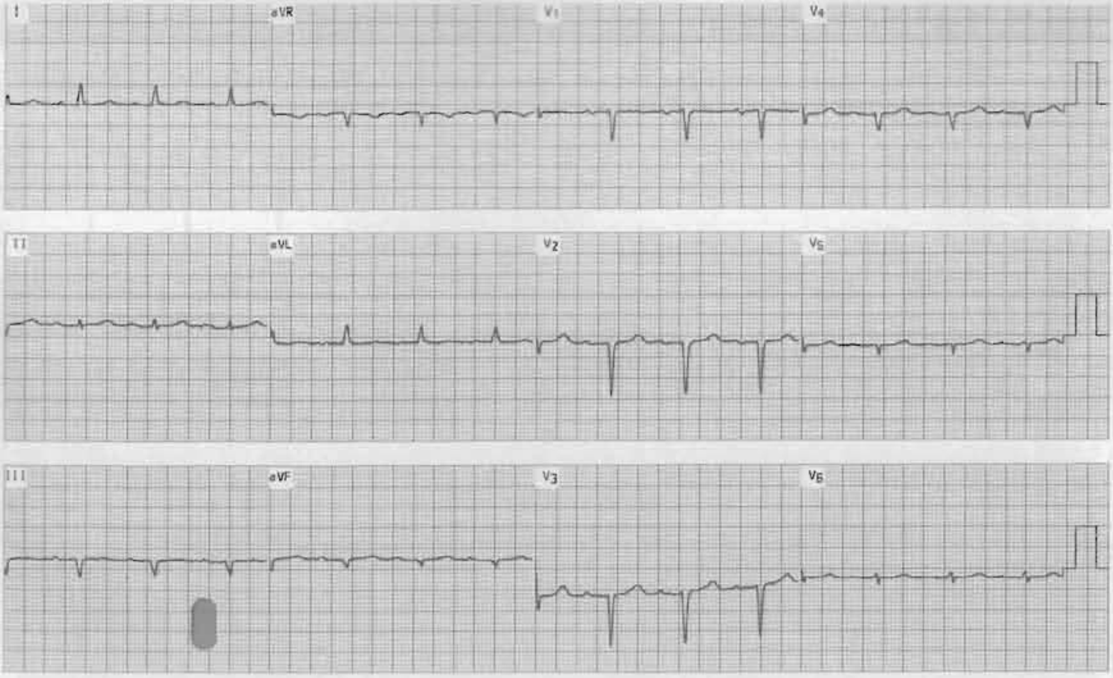


FIGURE 9.9 Hypertrophy present without ECG evidence.

shown in **Figure 9.9** had significant hypertrophy of the left ventricle as determined by transesophageal echocardiography, but the cardiogram does not meet any of the criteria for LVH. This is why terms like *voltage for LVH* are preferred. Such terms indicate the presence of an ECG finding without asserting the definite presence of morphological changes (see **Table 9.1**).

TABLE 9.1

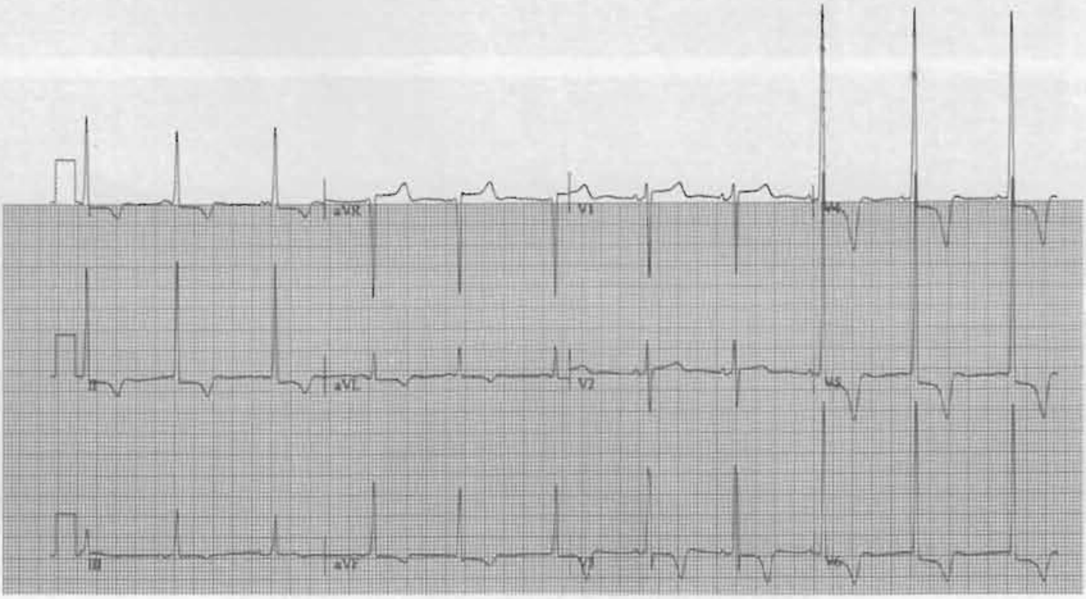
Hypertrophy Criteria

RAA	P wave in lead II height ≥2.5 mm
LAA	P wave in Lead II width ≥ 120 ms and/or negative part of P wave in Lead V_1 1 mm wide and 1 mm deep
RVH	R in V_1 ≥ S in V_1 RAD, persistent S waves in V_6 RAA
LVH*	R wave in Lead V_5 or V_6 + S wave in V_1 ≥ 35 mm and/or R wave in Lead AVL ≥12 mm and/or R wave in Lead I + S wave in Lead III > 25 mm

*Many different criteria for LVH have been described; we list three common ones. It should be recognized that other criteria may be used.

CLINICAL VIGNETTE REVISITED

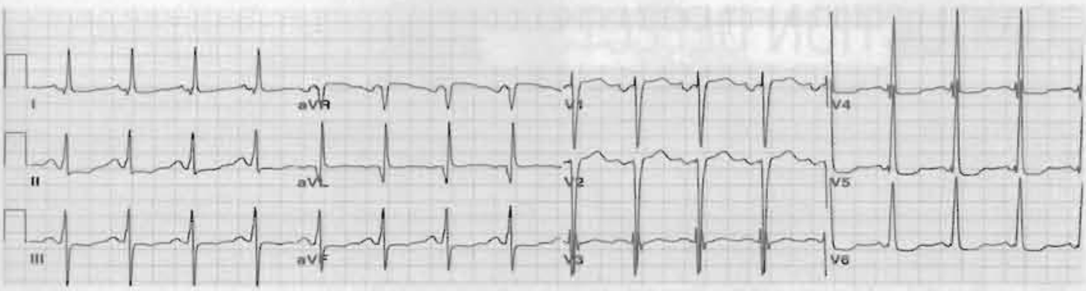
A patient presenting for a plain stress test has inverted T waves in numerous leads on the resting ECG. Should any extra steps be taken before placing him on the treadmill?



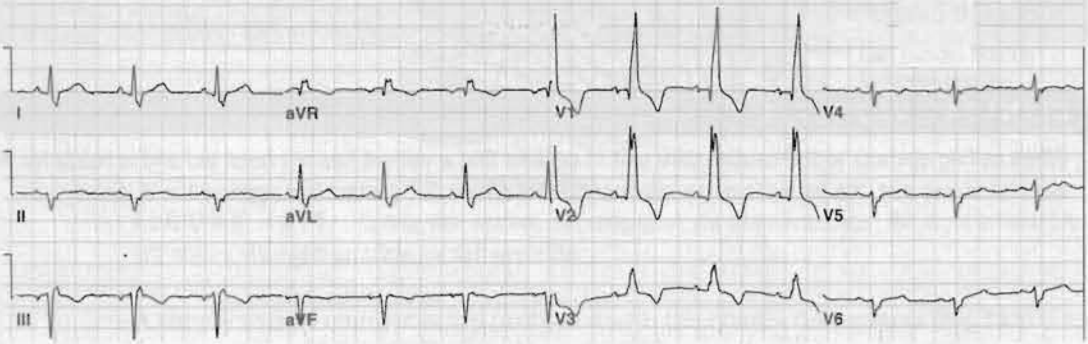
The T wave inversions appear to be repolarization abnormalities ("strain" pattern) associated with LVH. These changes make diagnosis of ischemia from the ECG alone difficult. The exercise specialist consulted with the attending cardiologist and the test was changed to a nuclear exercise stress test.

Quiz 9

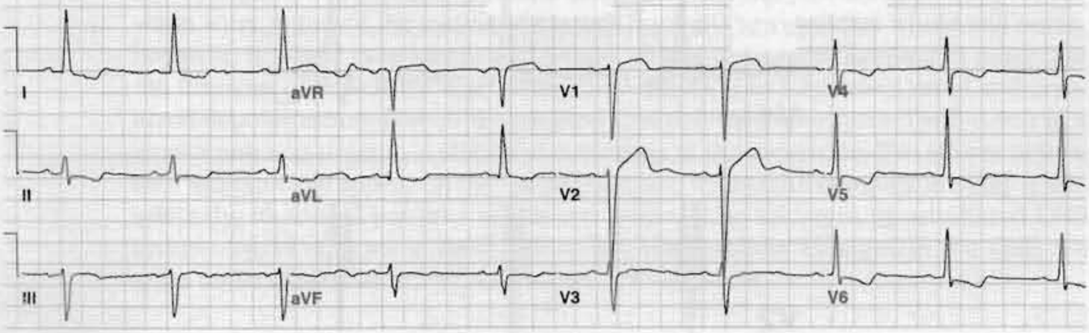
1. Describe any evidence of hypertrophy for the cardiograms below (A through C).


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6

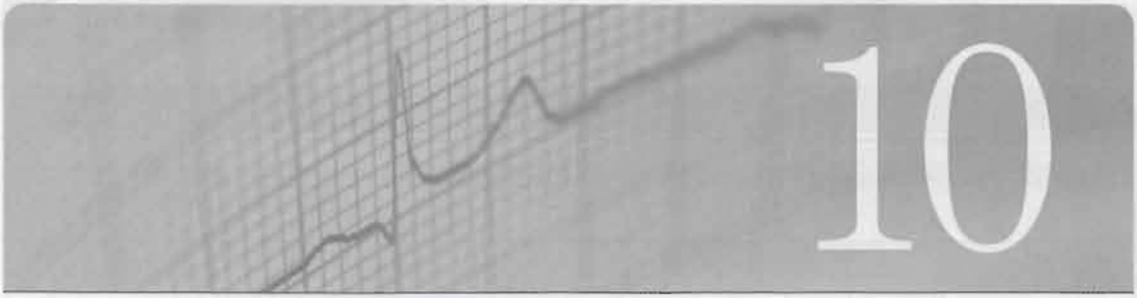
A


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6

B


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6

C



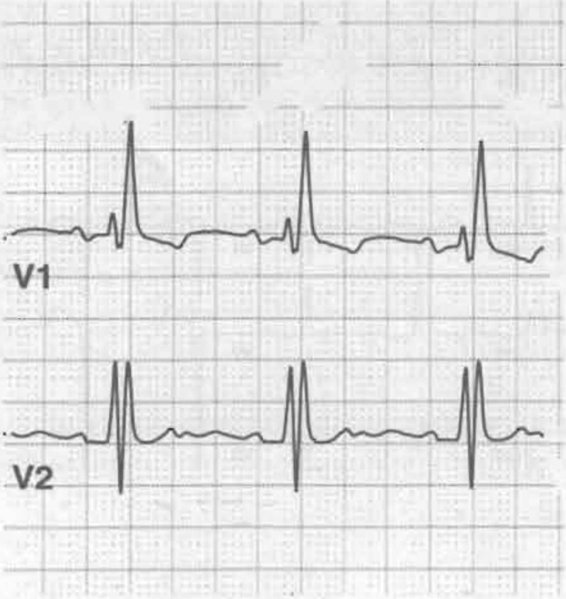
10

CONDUCTION DEFECTS

- Right Bundle Branch Block
- Left Bundle Branch Block
- Complete and Incomplete Bundle Branch Blocks
- Secondary ST-T Changes from Bundle Branch Blocks
- Rate-Related (Exercise-Induced) Bundle Branch Blocks
- Intraventricular Conduction Defects
- Fascicular (Hemi)blocks
- Bifascicular Blocks

CLINICAL VIGNETTE

When calling upstairs to the cardiac care unit to confirm that a patient has not been fed and is ready for his exercise stress test, the patient's nurse informs you that a new rSR′ pattern has appeared in V_1 and V_2.



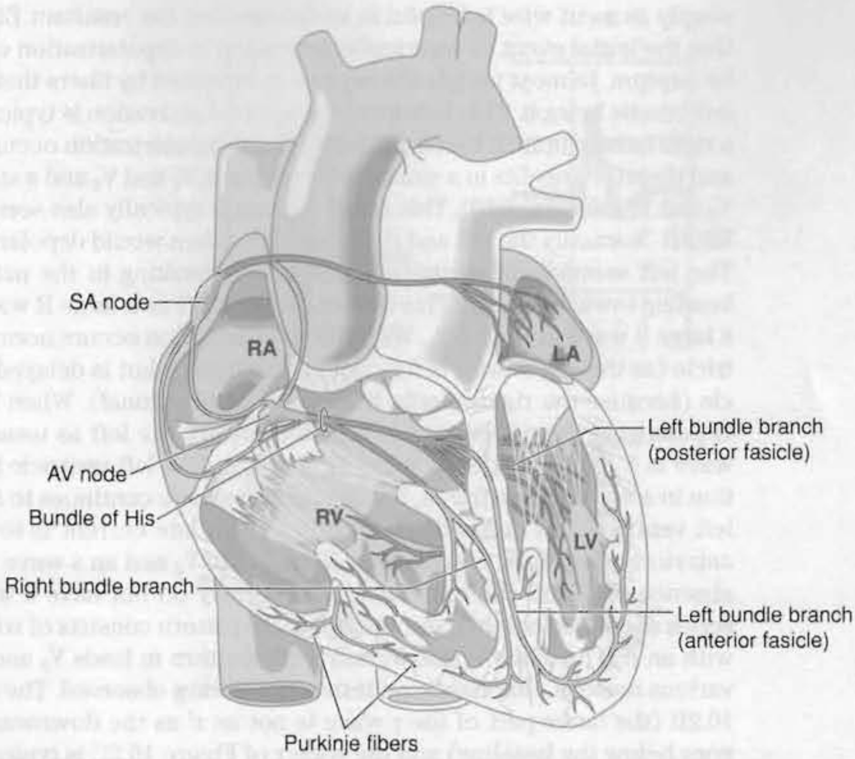

SA node
RA
LA
Left bundle branch (posterior fasicle)
AV node
Bundle of His
RV
LV
Right bundle branch
Left bundle branch (anterior fasicle)
Purkinje fibers

FIGURE 10.1 Conduction system. SA, sinoatrial; AV, atrioventricular; RA, right atrium; LA, left atrium; RV, right ventricle; LV, left ventricle.

A simple way to conceptualize problems with the conduction system **(Fig. 10.1)** is to imagine the right and left bundle branches as wires. Since the cells of the ventricular myocardium are linked together electrically by gap junctions, if one of these wires were to be cut the spread of current through the ventricles would still occur, however, it would take longer and would be less organized. Keeping in mind that the function of the ventricular conduction system is to facilitate a rapid and organized depolarization, it should be logical that defects in the system will result in a slower and less organized electrical activation of the ventricles. The slower activation will typically appear on the cardiogram as a wider QRS complex in many leads, while the abnormal patterns of current flow will be apparent by an altered QRS morphology. Using some basic principles, the resultant changes in the QRS are quite understandable.

Right Bundle Branch Block

Imagine that the left bundle branch is functioning normally, but the right bundle branch has somehow been "cut." In actuality, more commonly part of the system may be ischemic or have suffered degenerative changes, however, thinking of it simply as a cut wire is helpful in understanding the resultant ECG patterns. Recall that the initial event of ventricular activation is depolarization of the intraventricular septum. In most people the septum is activated by fibers that originate from the left bundle branch. This being the case, septal activation is typically not affected by a right bundle branch block (RBBB). Septal depolarization occurs from left to right, and therefore results in a small r wave in leads V_1 and V_2 and a small q wave in leads V_5 and V_6 (see Fig. 7.10). This initial pattern is typically also seen in the presence of RBBB. Normally the left and right ventricles then would depolarize simultaneously. The left ventricle is electrically dominant, resulting in the net vector of current heading toward the left. This is seen on the ECG as a large R wave in V_5 and V_6 and a large S wave in V_1 and V_2. With RBBB conduction occurs normally in the left ventricle (as the left bundle is functioning normally), but is delayed in the right ventricle (because the right bundle branch is not functional). When both ventricles are depolarizing the net current is directed toward the left as usual, resulting in an S wave in V_1 and V_2 and an R wave in V_5 and V_6. The left ventricle finishes depolarization in a normal time frame, but the right ventricle continues to depolarize after the left ventricle has finished. The vector of this late current is toward the right and anteriorly, therefore, results in an R' in V_1 and V_2 and an s wave in V_5 and V_6 (in the absence of RBBB the left chest leads usually do not have s waves). Putting this series of events together, the classic RBBB pattern consists of wide QRS complexes with an rSR' in leads V_1 and V_2 and a qRs pattern in leads V_5 and V_6 **(Fig. 10.2A)**. For various reasons, this classic pattern is not always observed. The notched r of Figure 10.2B (the latter part of the r wave is not an r' as the downward deflection never goes below the baseline) and the wide r of Figure 10.2C is typical of other patterns sometimes seen with RBBB. Note that in all cases the QRS is wide and the R waves are larger than would normally be seen in the right chest leads.

Left Bundle Branch Block

Imagine that the right bundle branch is functioning normally, but the left bundle branch has somehow been "cut." The right ventricle should depolarize quickly and normally, but the left ventricle will be activated by the slower cell to cell pathways. Also, septal activation will usually be affected as the septum is usually depolarized by fibers that originate from the left bundle branch. Therefore, the normal initial (septal) r waves in V_1 and V_2 will be lost as will the normal initial (septal) q waves of V_5 and V_6. Instead of the septum activating first, the initial event will be depolarization of the right and left ventricles, with the net vector of this current directed toward the more massive left ventricle. Because of the direction of current flow this will be seen on the ECG as an R wave in leads V_5 and V_6 and a Q wave in leads V_1 and V_2. The right ventricle will finish depolarizing in a normal amount of time, but the completion of left ventricular depolarization will take longer than usual, causing a continued (wide) R wave in the left chest leads. The resultant pattern of a left bundle branch block (LBBB) therefore is typically a wide (sometimes notched) R wave in leads V_5 and V_6 and a wide QS pattern in leads V_1 and V_2 **(Fig. 10.3)**. Leads I and aVL also typically show wide R waves as these leads are also leftward.

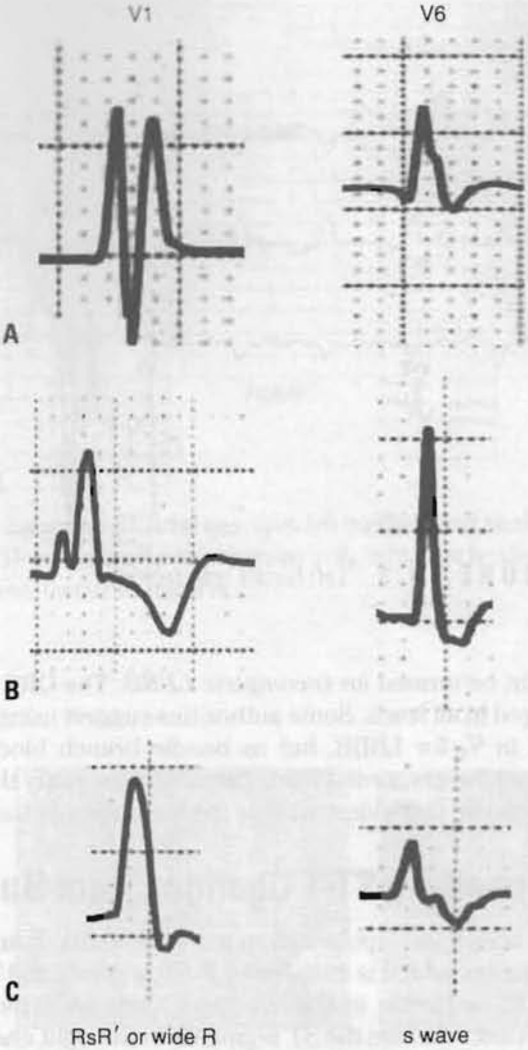


FIGURE 10.2 Right bundle branch block patterns.

Complete and Incomplete Bundle Branch Blocks

In both RBBB and LBBB ventricular depolarization takes longer than normal because part of the conduction system is disabled. This prolongs the QRS complexes in certain leads. A normal QRS duration is <100 ms (2.5 small boxes). If the QRS duration during a bundle branch block is ≥100 but <120 ms it is often called an *incomplete bundle branch block*, whereas if the QRS duration is ≥120 ms a *complete bundle branch block* is said to be present. For example, if a QS is seen in V_1 and a wide R in V_6 (LBBB pattern) and the QRS duration is slightly >100 ms this

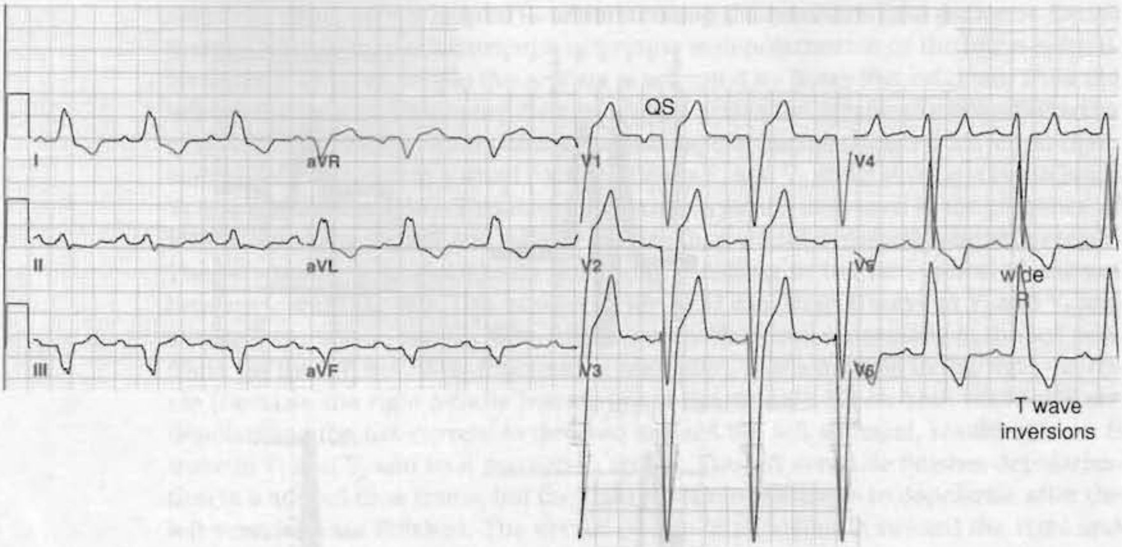


FIGURE 10.3 Left bundle branch block.

might be termed an *incomplete LBBB*. The QRS duration will usually not be prolonged in all leads. Some authorities suggest using the QRS duration in V_1 for RBBB and in V_6 for LBBB, but as bundle branch blocks were actually first described before the precordial leads came into use (only the limb leads existed at the time), others use the widest QRS of the limb leads as the benchmark.

Secondary ST-T Changes from Bundle Branch Blocks

The abnormal depolarization patterns of RBBB and LBBB result in abnormal repolarization, which is manifested in ST segment and T wave abnormalities. With LBBB the ST segments in the left chest leads are typically depressed and the T waves inverted, whereas the ST segments in the right chest leads are typically elevated. In the presence of RBBB ST segment depressions and T wave inversions are seen in the right chest leads, while the left chest leads are typically unaffected (other than the presence of the s waves previously described). These changes are shown in **Figure 10.4** and are called *secondary ST-T changes* because they are secondary to the abnormal depolarization occurring because of the bundle branch blocks. In later chapters primary ST-T changes (e.g., from myocardial infarction) will be described.

Rate-Related (Exercise-Induced) Bundle Branch Blocks

Figure 10.5 shows an ECG (leads V_1 and V_5) from the same patient under three conditions: rest (Fig. 10.5A), exercise (Fig. 10.5B), and after recovery from exercise (Fig. 10.5C). The QRS complex in lead V_6 is not wide at rest or during recovery, but shows

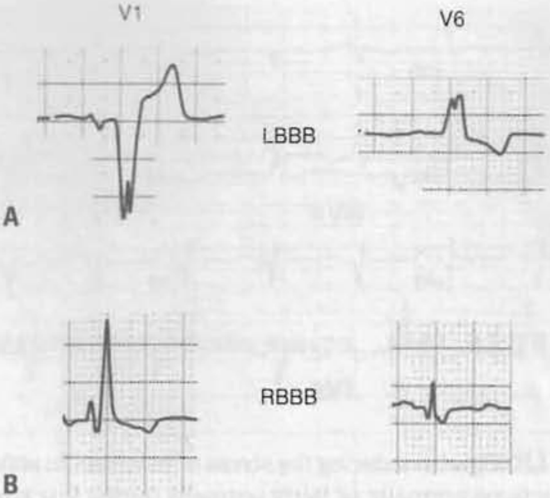


FIGURE 10.4 Secondary ST-T changes. With left bundle branch block (LBBB) (**A**), there is ST elevation in V_1 and ST depression/T wave inversion in V_6. With right bundle branch block (RBBB) (**B**), we see ST depression/T wave inversion in V_1.

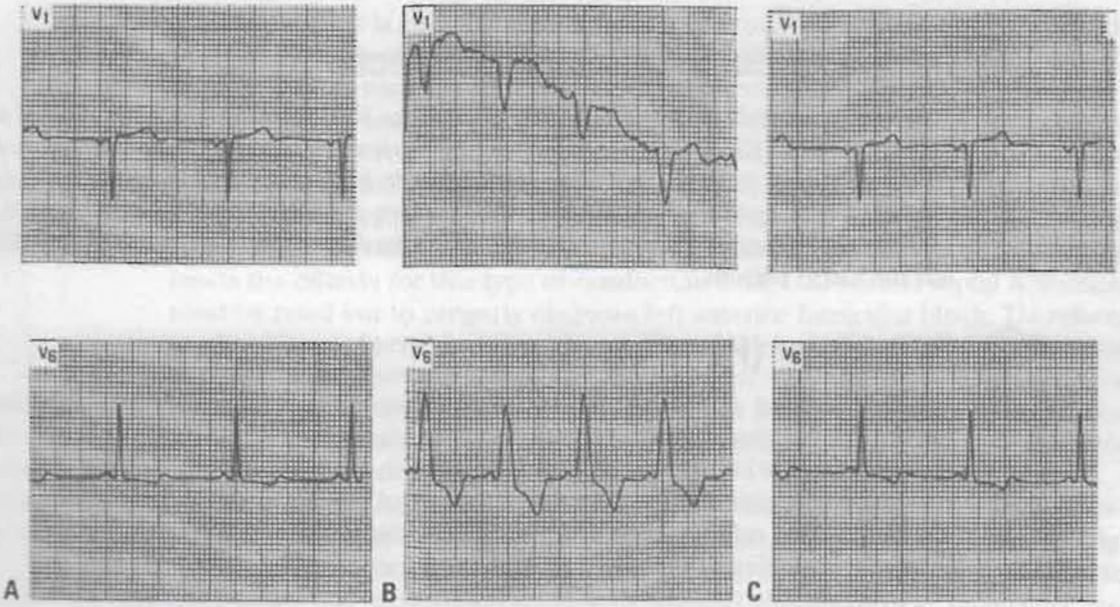


FIGURE 10.5 Rate-related left bundle branch block: at rest (**A**), with exercise (**B**), and during recovery (**C**).

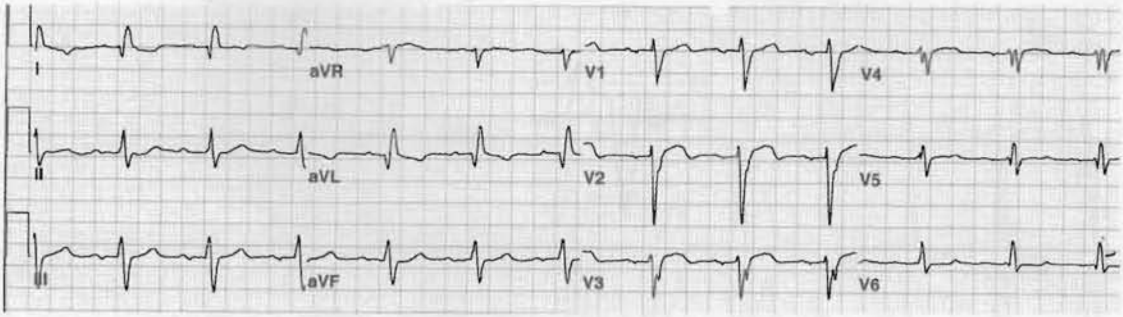


FIGURE 10.6 Intraventricular conduction defect (delay).

an LBBB pattern during the stress of exercise. In some patients, the conduction system functions normally or fairly normally during low stress conditions, but bundle branch blocks occur when the heart rate (HR) increases. Because of degenerative changes, ischemia, or other factors the additional stress of an increased HR leads to the temporary appearance of conduction defects. If this occurs during exercise this phenomenon is sometimes called an *exercise-induced bundle branch block*. Since these transient blocks coincide with increases in HR and may occur at rest if the HR is high enough, they may also be called *rate-related bundle branch blocks*. The V_1 exercise strip (Fig. 10.5B) also shows a type of artifact know as *wandering baseline*, which can be caused by patient movement.

Intraventricular Conduction Defects

Sometimes a conduction problem exists, but the ECG pattern is neither that of a RBBB nor a LBBB. In such cases the term *intraventricular conduction defect* (IVCD) or delay may be used. The QRS duration in **Figure 10.6** is abnormally long (i.e, ≥100 ms), however, neither the RBBB pattern nor the LBBB pattern is present. The use of the term IVCD recognizes a conduction defect that fits neither the RBBB nor the LBBB pattern.

Fascicular (Hemi)blocks

Although the left bundle branch is often spoken of as if it were one "wire," the human conduction system is better conceptualized as trifascicular, consisting of one right bundle branch and a left bundle branch with two major fascicles. Figure 10.1 shows the anterior and posterior fascicles of the left bundle branch. They are sometimes also referred to as *inferior* (for posterior) and *superior* (for anterior); the discussion here uses the anterior and posterior designations.

It is possible that one of these fascicles may be functioning normally while the other is not. Failure of one of the fascicles is called a *fascicular block* or *hemiblock*. Oddly, failure of one fascicle only mildly prolongs the QRS duration; a more

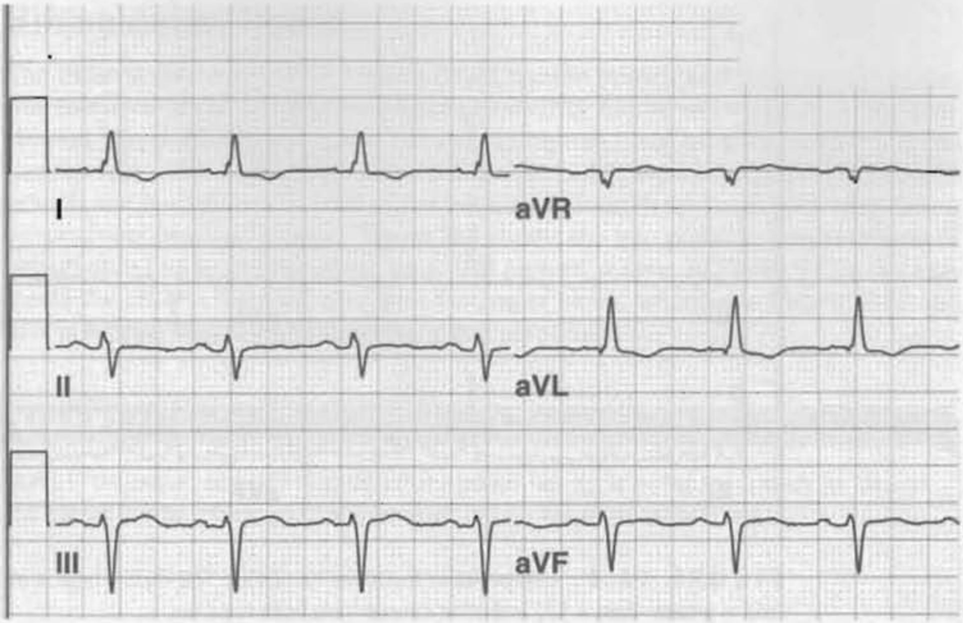


FIGURE 10.7 Left anterior fascicular hemiblock. Note that the axis is more negative than −45 degrees, and there is a Q wave in aVL.

significant effect is a shift of the axis. In the case of a left anterior fascicular block (also called left anterior hemiblock) the axis shifts to the left and becomes −45 degrees or more negative. Thus an axis of −45 or more negative (e.g., −60 degrees) is considered presumptive evidence of left anterior fascicular block. An easy way to recognize this is if the S wave in lead aVF is deeper than the R wave in lead I is tall. Notice that in **Figure 10.7** the S wave in lead aVF is about 7 mm deep, whereas the R wave in lead I is only about 5 mm tall. This is simply a quick method of recognition; the usual methods of axis determination will also reveal if the axis meets the criteria for this type of conduction defect. Abnormal septal activation must be ruled out to properly diagnose left anterior fascicular block. Therefore, small q waves of brief duration in two of the three leads I, aVL, and V_6 (evidence of normal septal activation) as well as an axis of −45 degrees or more negative must be present. The criteria for a left anterior fascicular block should not be applied in the presence on an inferior wall myocardial infarction as these infarcts shift the QRS axis.

If the left anterior fascicle of the left bundle is functioning normally but the posterior fascicle fails, the axis shifted well to the right and will be >120 degrees **(Fig. 10.8)**. Many factors can shift the axis to the right, so an axis >120 is only assumed to represent left posterior fascicular block when other causes (e.g., pulmonary disease) are ruled out. Therefore, the term *possible left posterior fascicular block* may be used to describe the finding of an axis >120 degrees.

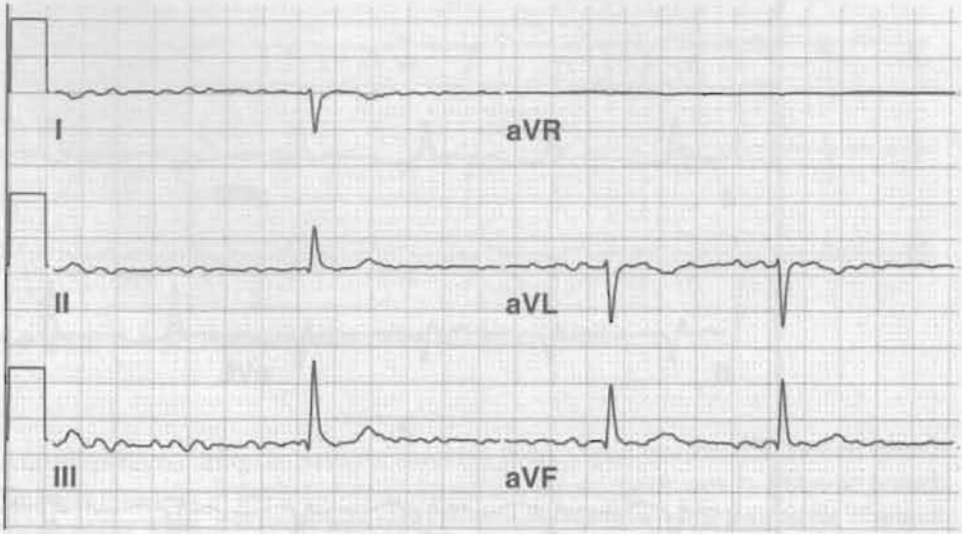

I
aVR
II
aVL
III
aVF

FIGURE 10.8 Left posterior fascicular hemiblock. The rhythm here is atrial fibrillation. The axis is greater than +120 and other causes have been ruled out.

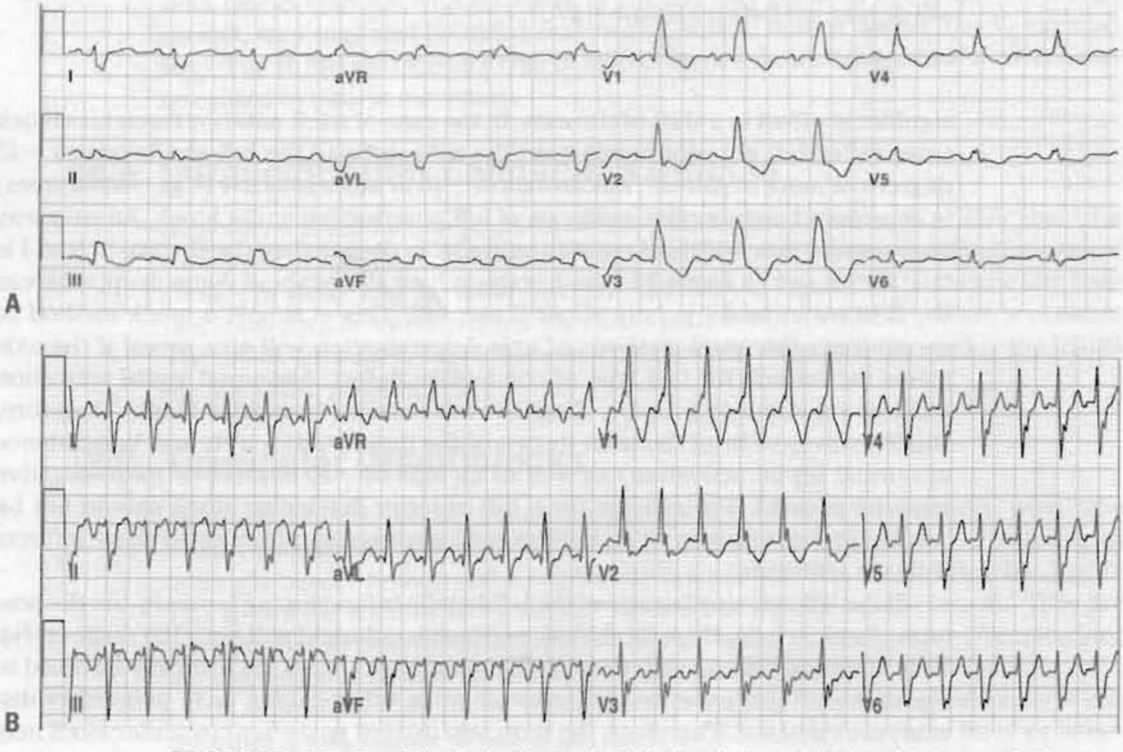

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
A
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
B

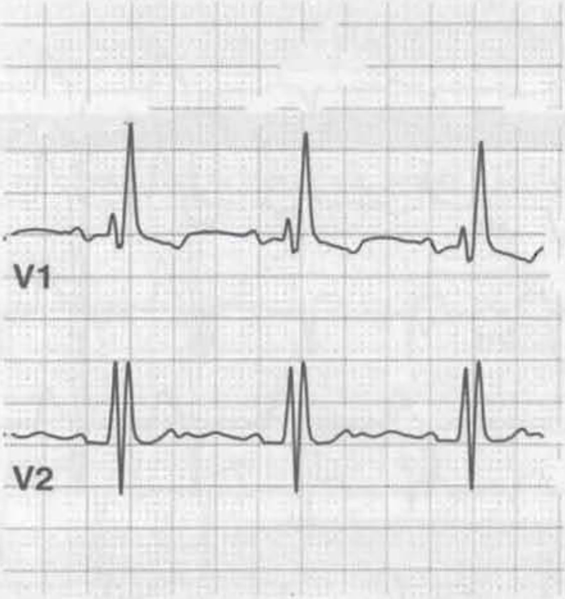
FIGURE 10.9 Bifascicular blocks. **A:** Right bundle branch block and left *posterior* fascicular block. **B:** Right bundle branch block and left *anterior* fascicular block.

KSA Bifascicular Blocks

The trifascicular conduction system (right bundle branch and anterior and posterior fascicles of the left bundle branch) can suffer failure of two of the three constituent pieces (if all three parts fail then complete heart block is present). If the two fascicles of the left bundle branch fail, then LBBB is present. If the right bundle branch and one of the fascicles of the left bundle branch are malfunctioning then a *bifascicular block* is present.* **Figure 10.9** shows the two possible combinations of bifascicular block. In both cases, an RBBB pattern is seen. In Figure 10.9A the axis meets the criteria for a left posterior fascicular block, whereas in Figure 10.9B the axis is representative of left anterior fascicular block.

CLINICAL VIGNETTE REVISITED

When calling upstairs to the cardiac care unit to confirm that a patient has not been fed and is ready for his exercise stress test, the patient's nurse informs you that a new rSR′ pattern has appeared in V_1 and V_2.



This patient has a new onset RBBB. Because this is a significant change in clinical status, the laboratory's medical director is contacted for instructions before the exercise test.

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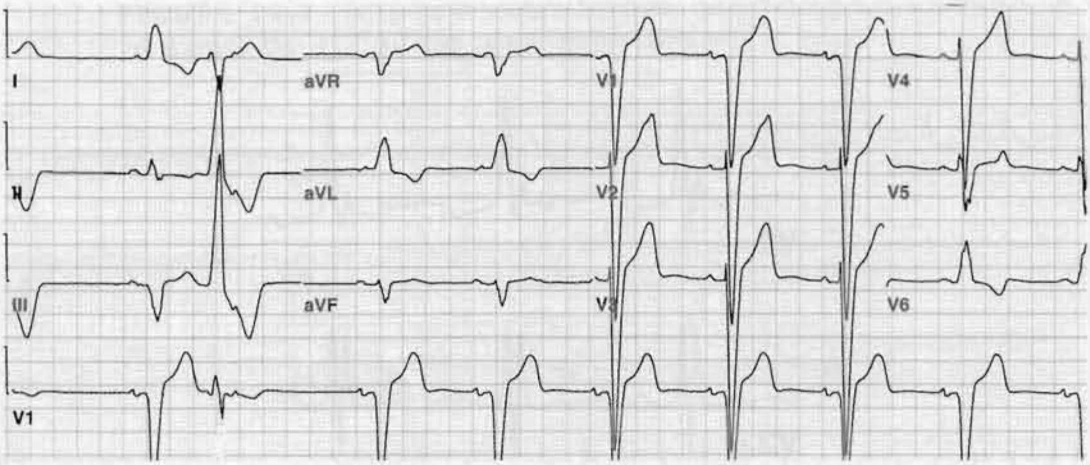
*Some clinicians also consider first degree AV block in combination with a bifascicular block to be a form of tri-fascicular block.

Quiz 10

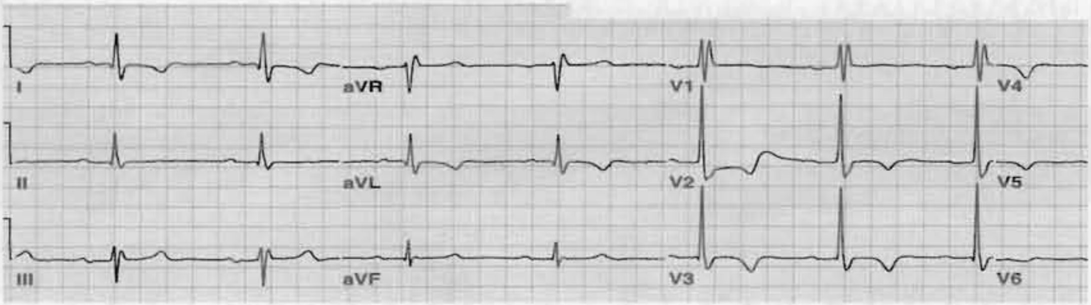
1. Describe the QRS patterns seen with RBBB and LBBB.

2. What is meant by the term intraventricular conduction defect (or delay)?

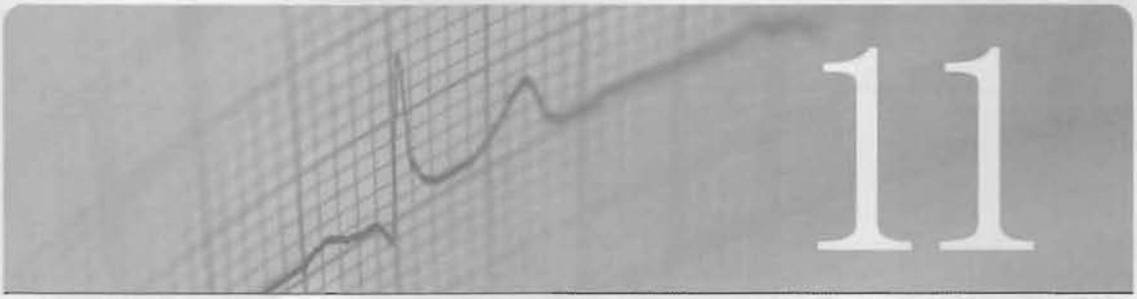
3. Describe any evidence of conduction defects for the cardiograms below (A and B).


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1

A


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6

B



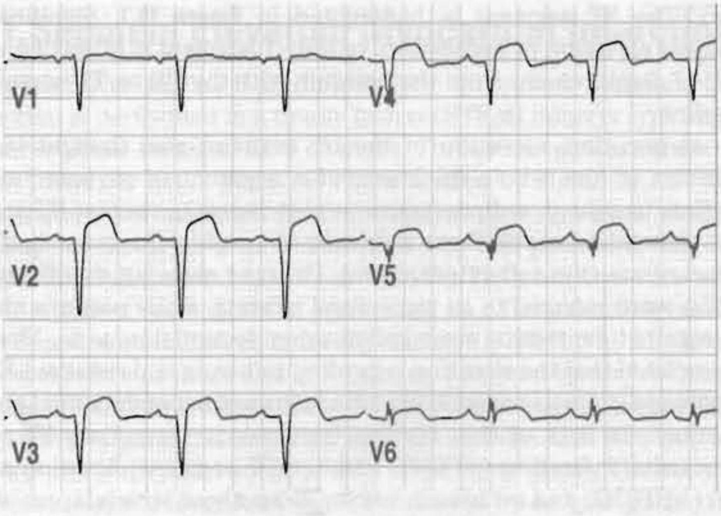
11

ISCHEMIA AND INFARCT

- ST Segment Elevation Myocardial Infarction
- Q Waves
- Indeterminate Age Infarct
- Non-ST Segment Elevation Infarction
- Localization of Infarcts
- "Reciprocal" Changes/Remote Ischemia
- Right Ventricular Infarctions
- Posterior Wall Infarctions
- Infarctions and Bundle Branch Blocks
- ST-T Wave Changes Not Caused by Ischemia/Infarct

CLINICAL VIGNETTE

A patient awaiting a stress test complains that he feels a strong pressure in his chest. He appears pale and is sweating profusely.



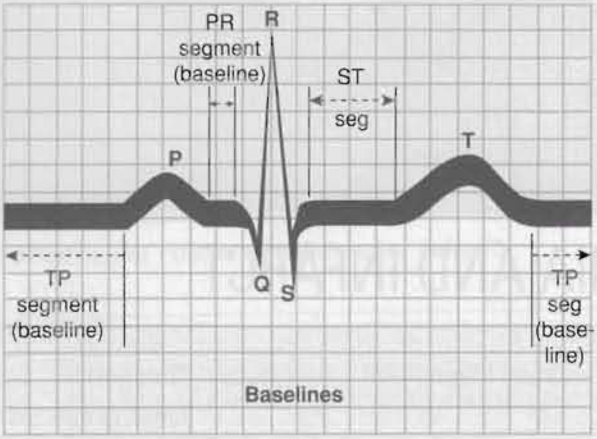


FIGURE 11.1 Isoelectric points.

Myocardial ischemia, a lack of oxygen, is usually due to an inadequate supply of blood, which in turn is usually due to blockage of a coronary artery. If prolonged, ischemia leads to increasing damage to and eventual death of myocardial tissue. This is termed a *myocardial infarction* or, in layman's terms, a "heart attack." Myocardial ischemia and infarction cause distinctive changes in the cardiogram, with many of these changes affecting the ST segment and/or the T wave. Recall that the ST segment begins at the end of the QRS complex and ends at the beginning of the T wave and that the normal ST segment should be isoelectric (i.e., on the baseline). The ST segment is highlighted in **Figure 11.1.** Significant displacement, whether elevation or depression, of the ST segment is often defined as ≥1 mm (0.1 mV) of displacement from the baseline with the PR or TP segment serving as the baseline.

At one time elevation of the ST segment was thought to be indicative of ischemia of the whole thickness of a segment of myocardium from subendocardium to subepicardium (transmural ischemia), whereas ST segment depression was thought to result from ischemia of roughly the inner half of a segment of myocardium (the subendocardium). Patterns showing significant ST segment elevation were referred to as transmural infarcts, while patterns showing significant ST segment depression were called subendocardial infarcts. More recently, it was appreciated that the situation regarding ischemia and resultant ECG changes does not always seem to coincide with the historical subendocardial and transmural designations. In light of this, current terminology recognizes ST segment elevation myocardial infarction (STEMI) and non-ST segment elevation myocardial infarction (NSTEMI) and avoids any assumptions about which layers of the myocardium are involved. It is currently thought that complete occlusion of a coronary artery results in ST segment elevation, whereas partial occlusion results in ST segment depression and/or T wave inversion. Some typical patterns of ST segment elevation and depression are shown in **Figure 11.2.**

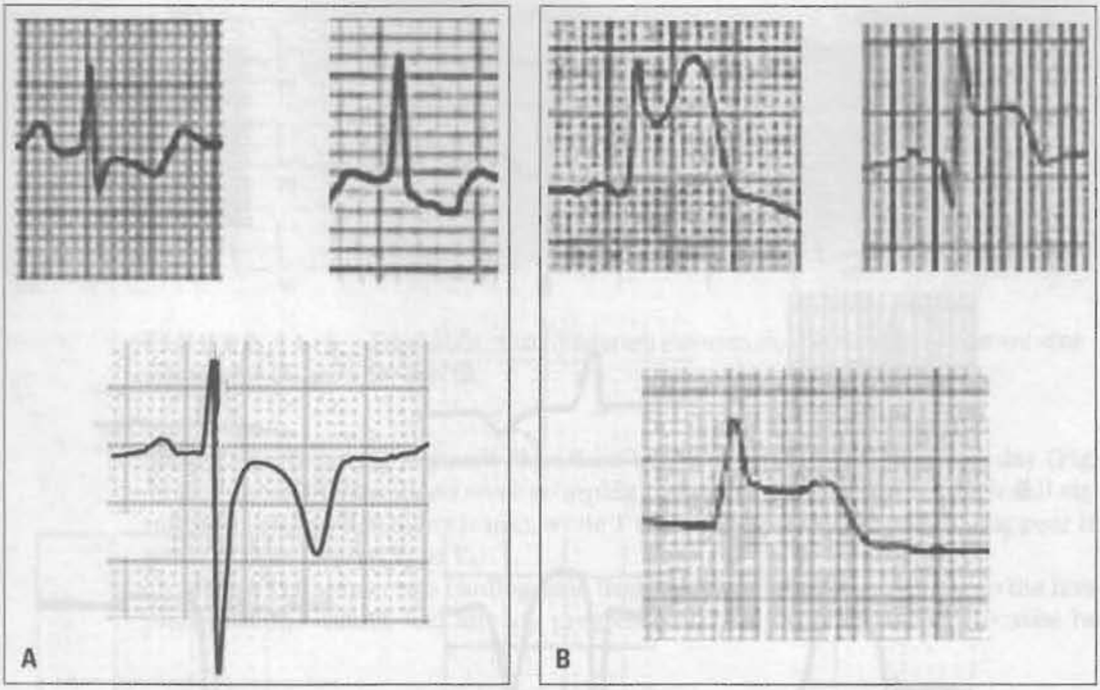


FIGURE 11.2 ST changes with ischemia. **A:** Partial occlusion showing ST depression and T wave inversion. **B:** Complete occlusion showing ST elevation.

ST Segment Elevation Myocardial Infarction

If no intervention such as thrombolytic therapy or percutaneous coronary intervention is performed in a timely fashion STEMI infarcts typically progress through a three-phase series of ECG changes. Three examples are shown in **Figure 11.3**: the first phase, or *acute phase*, is characterized by significant ST segment elevations sometimes accompanied by the onset of new Q waves. Actually the first ECG change of this type of infarct is often very tall, peaked T waves sometimes called *hyperacute T waves* (leads V_1 to V_3 in **Fig. 11.4**), but these are brief and often gone by the time a cardiogram is performed, so ST segment elevations are usually the first pattern encountered clinically during a STEMI. The ST elevations may last for several hours or more and are typically followed by an "evolving" pattern characterized chiefly by a return of the ST segments toward baseline (although still possibly elevated) and the inversion of T waves. New Q waves may also appear during this phase if they had not appeared in the acute phase. The *evolving phase* lasts for hours to days or longer and often is followed by an "old" pattern that primarily consists of Q waves. During the *old phase* the ST segments and T waves may have normalized, and the only evidence of the infarct remaining may be Q waves.

Figure 11.5 shows ECGs taken on the same patient during the acute and evolving phases of an STEMI. Notice that the first cardiogram (Fig. 11.5A)

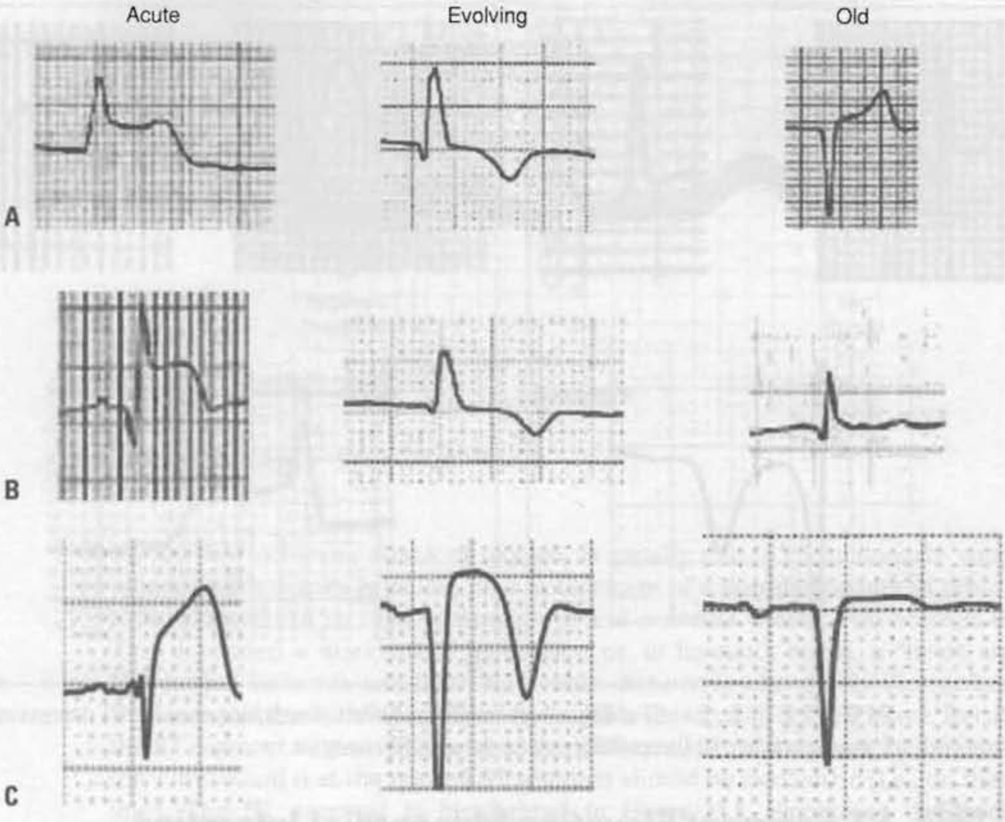

Acute
Evolving
Old
A
B
C

FIGURE 11.3 ST segment elevation myocardial infarction phases. **A, B, C:** Three examples of acute, evolving, and old phases of a STEMI.

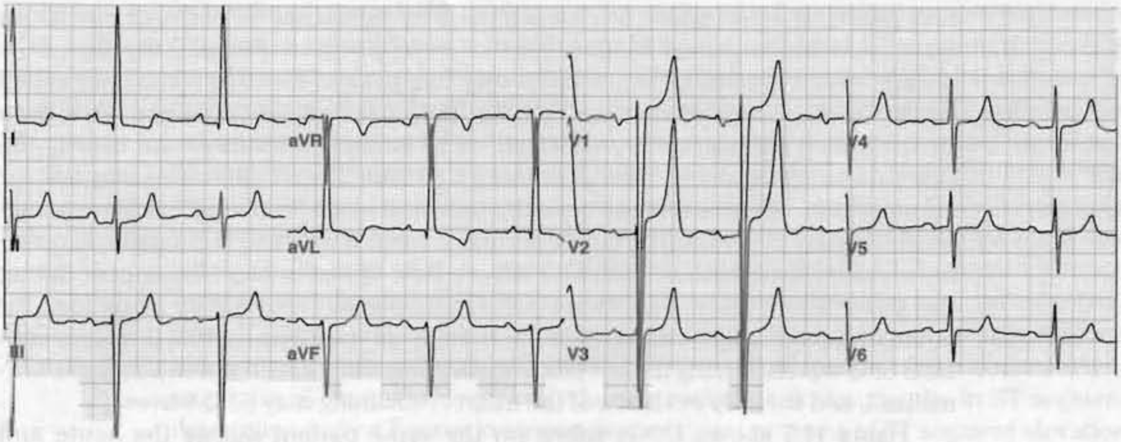

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6

FIGURE 11.4 Hyperacute T waves.

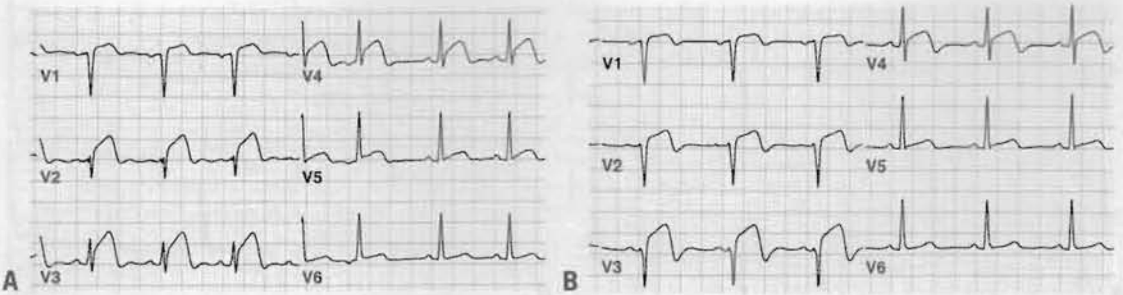

V1
V4
V2
V5
V3
V6
A
V1
V4
V2
V5
V3
V6
B

FIGURE 11.5 Serial ECGs of an ST segment elevation myocardial infarction patient upon presentation (**A**) and a day later (**B**).

shows significant ST segment elevations in the chest leads. By the next day (Fig. 11.5B), these ST elevations were returning towards the baseline (although still significantly elevated in many leads), while T wave inversions have begun to appear in some of these leads (V_2 to V_4).

Figure 11.6 shows two cardiograms from a patient who did not come to the hospital until his infarct had already progressed to the evolving phase. Because he

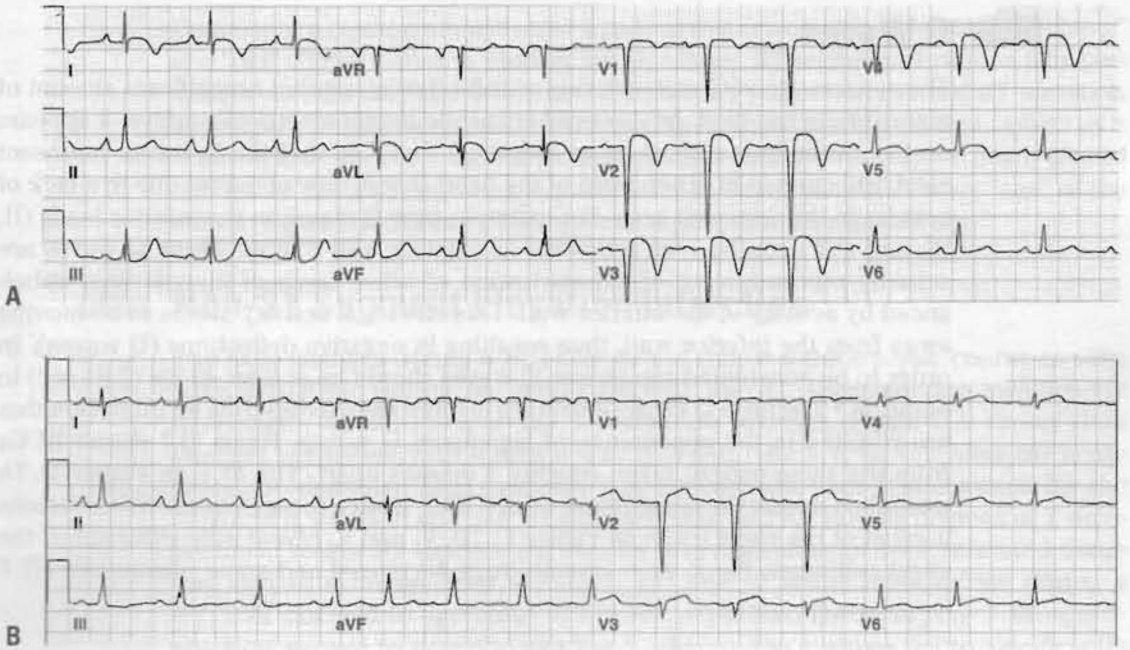

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
A
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
B

FIGURE 11.6 Late presentation ST segment elevation myocardial infarction (**A**) and 4 days later (**B**).

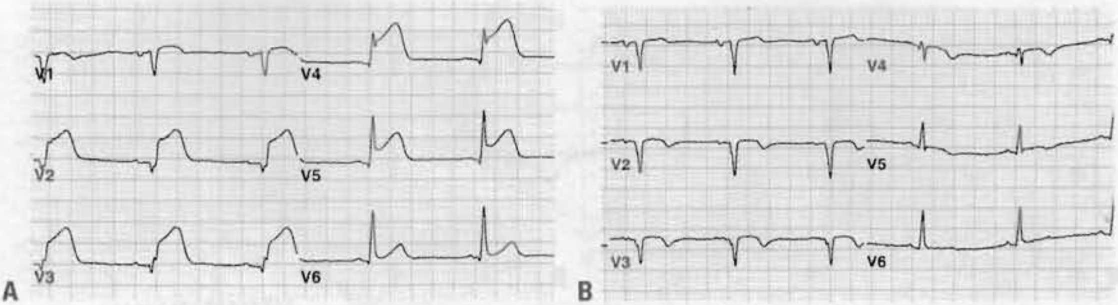


FIGURE 11.7 Acute and old stages of ST segment elevation myocardial infarction upon presentation (**A**) and 3 months later (**B**).

attributed his substernal sensation of pressure to "indigestion," this patient waited several hours before seeking medical attention. Presumably, an ECG taken earlier would have revealed large ST segment elevations in many of the chest leads. This can be inferred by the patterns of modest ST elevations combined with T wave inversions in V_1 to V_5 (which should come after the acute phase of very significant ST elevation). Four days later (Fig. 11.6B) some residual ST elevations remain, but the main abnormality is the presence of significant Q waves. In this case the T waves are no longer inverted.

Q Waves

The appearance of Q waves during an infarct suggests that a significant amount of myocardium has died. As this dead area is no longer electrically active, it appears on the cardiogram almost as an electrical "hole" in that the Q waves represent electrical current in other areas of the heart that is now apparent due to a lack of activity in the damaged area. For example, new Q waves in the inferior leads (II, III, and aVF) can be seen following an inferior wall STEMI. These Q waves are actually representative of depolarization of other areas of myocardium unbalanced by activity of the inferior wall. This electrical activity seems to be moving away from the inferior wall, thus resulting in negative deflections (Q waves). In order to be considered significant, Q waves should be at least 40 ms (0.04 sec) in duration.* The term *Q wave infarction* is often used to describe an infarction that has resulted in the appearance of significant Q waves. **Figure 11.7** shows ECGs from the same patient taken roughly 3 months apart. The ECG in Figure 11.7A was taken during the acute phase of a STEMI. Notice the significant ST elevations in most of the chest leads. In Figure 11.7B, V_5 and V_6 reveal little evidence of the event (although the R wave amplitude is decreased and some nonspecific ST-T

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*Other criteria for "significant" Q waves have been described; we present one simple commonly used criterion.

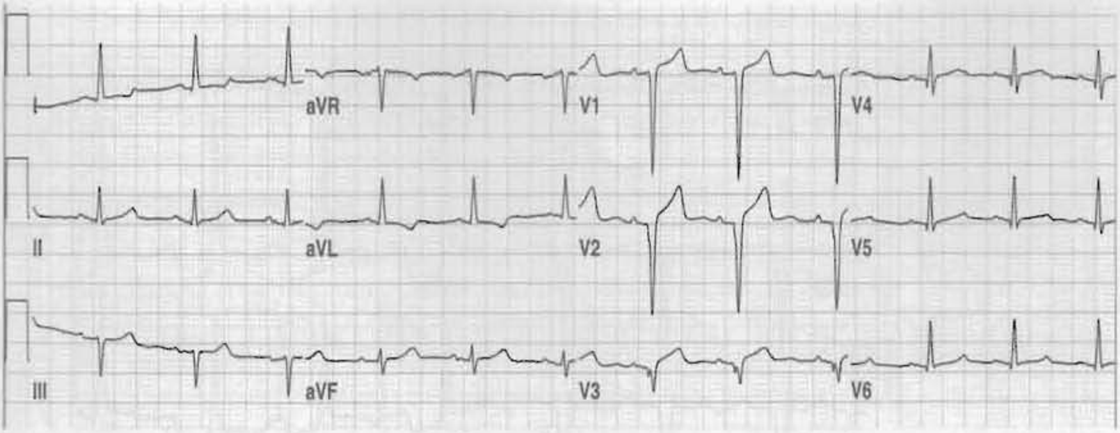


FIGURE 11.8 Indeterminate age infarct.

wave changes are present), while V_1 to V_4 show the telltale Q waves, indicating an "old" STEMI.

Indeterminate Age Infarct

Commonly, a cardiogram will show a pattern that is a combination of the "evolving" and "old" STEMI patterns. Modest but significant ST segment elevations may persist for months or years following an infarct. A pattern of residual ST elevation accompanied by Q waves is often referred to as an *indeterminate age infarct* as it is a hybrid of the evolving and old patterns and therefore cannot be neatly placed into either category, resulting in an inability to determine the relative "age" of the infarct. **Figure 11.8** (leads V_1-V_3) shows an infarct of indeterminate age.

Non-ST Segment Elevation Infarction

The ECG changes during a NSTEMI do not follow a three-phase course as seen with a STEMI. During an acute NSTEMI the ST segments are significantly depressed (≥1 mm) and/or the T waves are inverted. **Figure 11.9** shows ECG changes consistent with a lateral wall NSTEMI (leads V_4 to V_6). After the acute phase, no definitive ECG evidence may remain, although it is common for the height of R waves in affected areas to decrease and for varying degrees of T wave inversion and/or ST segment depression to remain. Just as dead, infarcted tissue can lead to Q waves with a STEMI, the loss of viable myocardium during a NSTEMI can lead to decreases in R wave amplitude. The term *poor r wave progression* is used to describe situations wherein the r waves fail to significantly increase in amplitude across the chest leads. This can be caused by an infarct or can occur from other causes.

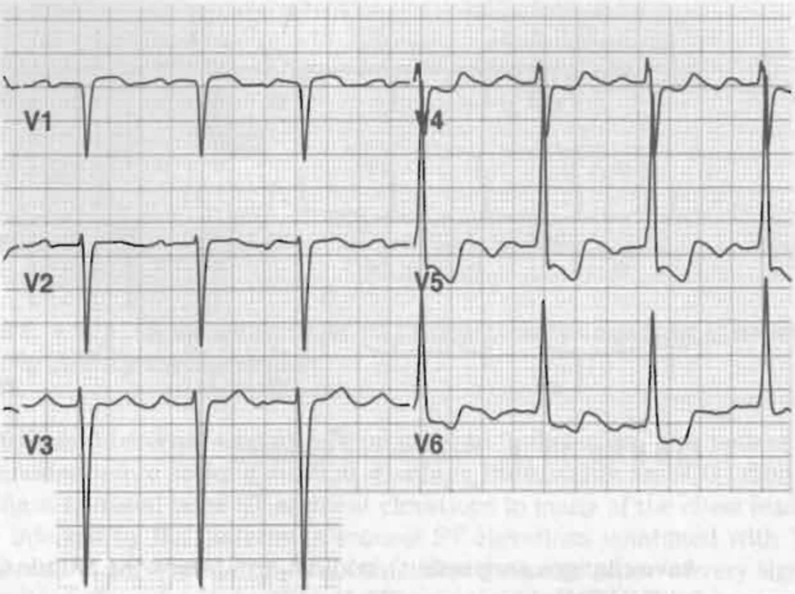


FIGURE 11.9 Non-ST segment elevation myocardial infarction.

Comparison with previous ECGs may be useful to ascertain whether such findings are new and thus presumably due to a NSTEMI or were preexisting. **Figure 11.10** shows two ECGs from the same patient. The ECG shown in Figure 11.10B might was viewed as suspicious for acute ischemia/infarction (possible NSTEMI). The ECG in Figure 11.10A was taken during a NSTEMI, which occurred 5 years previously. The availability of the previous ECG (Fig. 11.10A) assisted in determining that the ST-T changes seen in Figure 11.10B were likely preexisting and not caused by an acute ischemic event.

If no previous ECGs are available, and sometimes even if they are, it may be difficult or impossible to diagnose a NSTEMI from the cardiogram after it has resolved. This is in contrast to STEMI postinfarct Q waves, which can persist for years and sometimes for life, allowing diagnosis of a previous STEMI long after the event.

Localization of Infarcts

Recall that different leads are positioned to electrically "view" different areas of the heart (Chapter 7). A given myocardial infarction typically affects only one or two areas of the heart; therefore, ECG changes associated with an infarct are often confined to a few leads. For example, a lateral wall STEMI results in ST segment elevations in leads V_5 and V_6 and/or I and aVL, whereas an inferior wall NSTEMI causes ST depression and/or T wave inversions in leads II, III, and aVF. Combinations of areas can also be

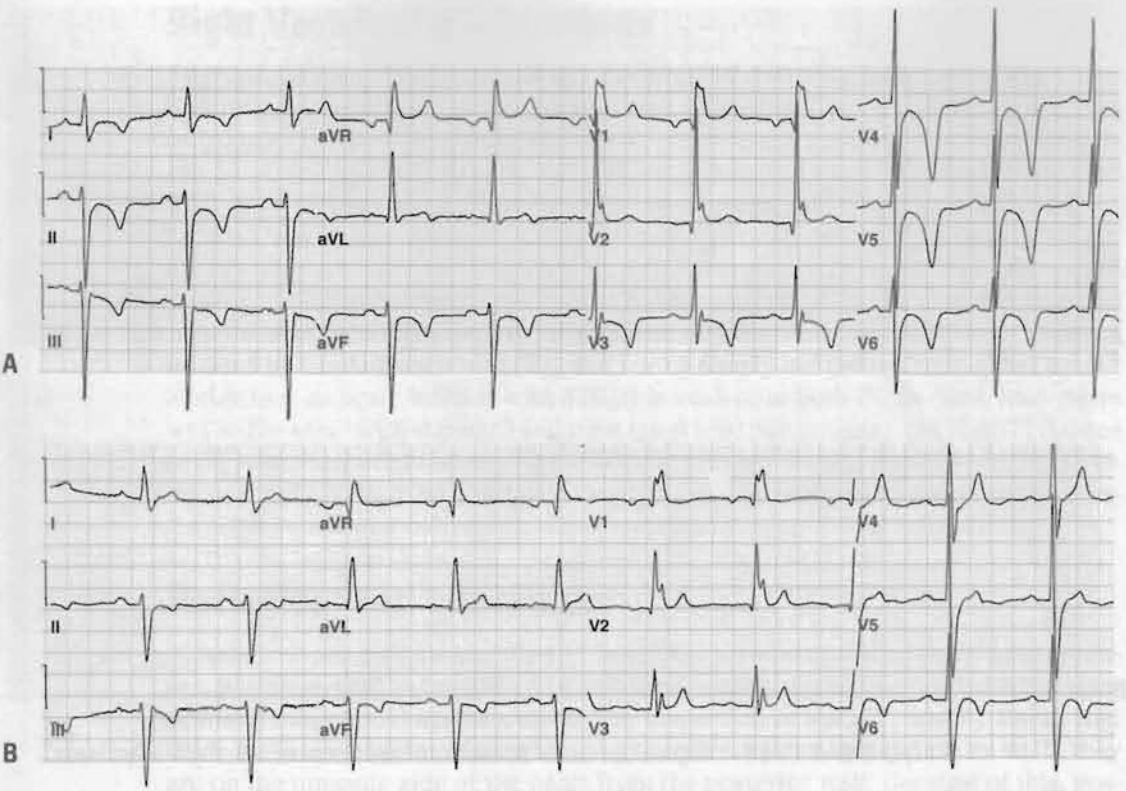


FIGURE 11.10 **A:** A patient with non-ST segment elevation myocardial infarction. **B:** The same patient, 5 years later.

described. For example, significant ST segment elevations in V_1, V_2, V_3, and V_4 would be indicative of an anteroseptal STEMI (anterior and septal walls); another patient with ST segment depressions and T wave inversions in II, III, aVF, V_5, and V_6 could be said to be suffering an inferolateral NSTEMI (inferior and lateral walls).

TABLE 11.1

Leads Associated with Area of Infarct

Area	Leads
Inferior	II, III, aVF
Lateral	I, aVL, V_5, V_6
Septal	V_1, V_2
Anterior	V_3, V_4
Posterior	V_1, V_2 (mirror image)

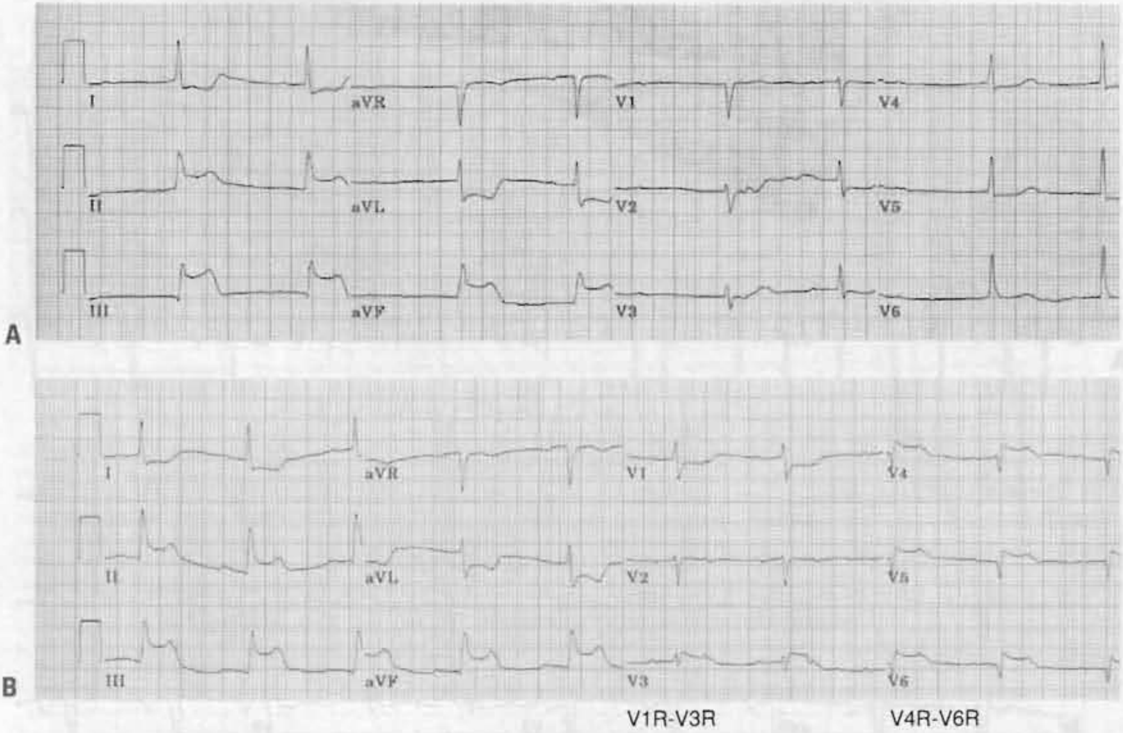


FIGURE 11.11 Right-sided leads. **A:** Standard lead placement. **B:** Right-sided leads.

"Reciprocal" Changes/Remote Ischemia

Sometimes during a STEMI (which by definition is associated with ST segment elevations) ST segment depressions are seen in a different area of the heart. **Figure 11.11A** reveals an inferior wall STEMI (with right ventricular involvement as shown in Fig. 11.11B) with typical ST elevations in the inferior leads. Note that the ST segments are depressed in leads I and aVL of the same ECG. It had been believed that such ST depressions seen in an area distant from the ST elevations were simply a sort of mirror image of the STEMI. In other words, the same area of ischemia/infarct (inferior wall in this example) would appear as ST elevations in leads covering the affected area and ST depressions in leads focused on the "opposite" side of the heart (the lateral wall in this case). This concept has generally been discredited and it is now believed that ST depressions occurring during a STEMI are actually representative of ischemia in another area of the heart. In this model, the ST elevations seen in leads II, III, and aVF of Figure 11.11 are indicative of an inferior wall STEMI, and the ST depressions in leads I and aVL represent ischemia of the lateral wall (sometimes referred to as "remote" ischemia as it is distant from the site of the infarct). The theory is that changes in myocardial function and therefore myocardial perfusion caused by an infarct in one area of the heart can cause another area of the heart to become ischemic.

Right Ventricular Infarctions

Although the septal leads (V_1 and V_2) are often referred to as "right" chest leads, this is in relation to the left ventricle. A standard 12-lead ECG has very limited coverage of the right ventricle. If a right ventricular infarction is suspected, additional leads can be placed on the right side of the chest. Figure 11.11 shows a standard 12-lead ECG (Fig. 11.11A) and an ECG with the chest leads shifted to the right side (Fig. 11.11B). For a right-sided ECG the normal V_2 becomes lead V_{1R}, the normal V_1 become V_{2R}, and the electrodes for V_{3R} to V_{6R} are placed in the normal anatomical locations, but on the right rather than the left side of the chest. The ECG should then be labeled in some way to make it clear that this placement is used. This can be done by labeling each lead (V_{1R}, etc.) or by simply indicating "right-sided leads." Notice that an acute inferior wall STEMI is evident in both ECGs (limb lead views will be the same with standard and right chest lead placements), but the ST changes of the concurrent right ventricular infarction are only seen in Figure 11.11B (ST elevations in V_{3R} to V_{6R}). With the usual anatomical distributions of the coronary arteries, right ventricular infarctions often accompany inferior wall infarctions.

Posterior Wall Infarctions

Although no leads of the standard 12-lead ECG are positioned on the posterior thorax, *posterior wall infarctions* (for historical reasons sometimes called true posterior wall infarctions) can be detected by inspection of leads V_1 and V_2. Recall that these leads are positioned roughly over the intraventricular septum. As such, they are on the opposite side of the heart from the posterior wall. Because of this, posterior wall infarctions are seen as a mirror image in V_1 and V_2. **Figure 11.12** shows two examples (Fig. 11.12A and Fig. 11.12B) of posterior wall infarcts as seen in a septal lead. The left-hand images show the ECG as it actually appears in the septal lead, while the images on the right are mirror images. Notice that the mirror images show Q waves and ST segment elevations consistent with a STEMI. In order to appreciate these changes on the original ECG one could (a) view leads V_1 and V_2 in a mirror (with the mirror at a 90-degree angle to the paper); (b) flip the ECG over, hold it up to a light, and read it through the paper; or (c) imagine the tracing as it would appear upside down (R waves imagined as Q waves, ST depressions pictured as ST elevations, etc.). If relatively large R waves appear in V_1 and/or V_2 one of these techniques should be used to search for a posterior wall infarct. As the normal pattern in these leads is a small r and a large S wave, an R wave that is about the same size as the s wave or larger is considered relatively large. Therefore, an r wave of only a few millimeters (Fig. 11.12B) could be considered "large" in the septal leads.

Infarctions and Bundle Branch Blocks

Recall from Chapter 10 that left bundle branch blocks usually result in T wave inversions and ST segment depressions in the left chest leads and Q waves and ST elevations in the right chest leads, whereas right bundle branch blocks cause ST

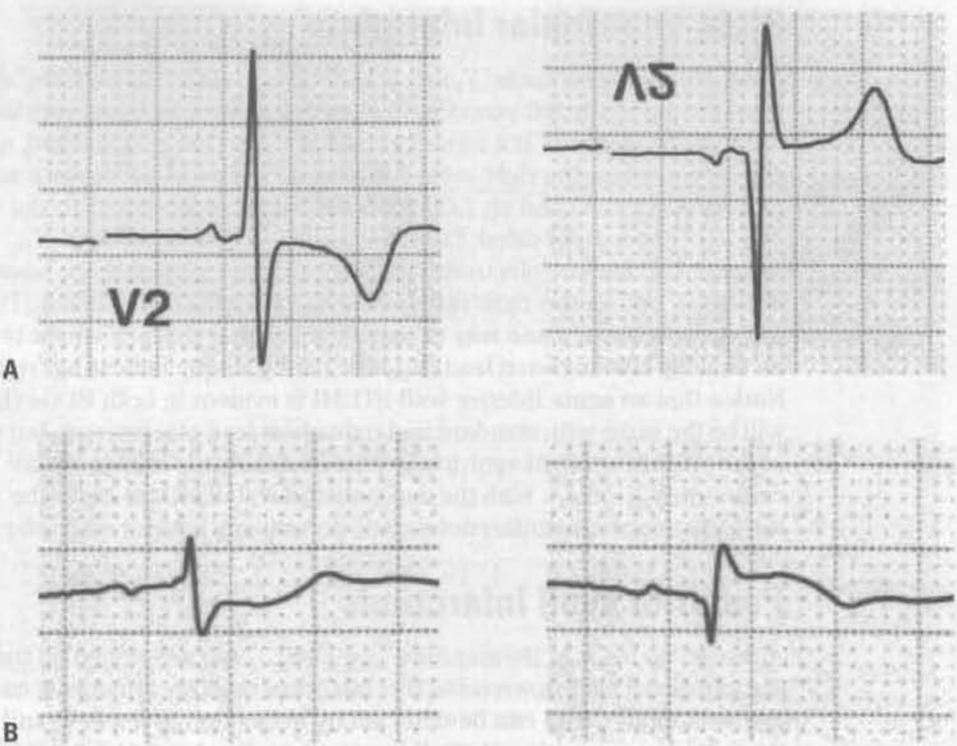


FIGURE 11.12 Posterior wall infarct. This figure shows the septal lead appearance (*left*) and the mirror image (*right*). **A** and **B** are two examples.

depressions and T wave inversions in the right chest leads. Such changes are often referred to as secondary ST-T changes because they are secondary to the abnormal depolarization and repolarization patterns associated with bundle branch blocks. These changes present a challenge when attempting to diagnose infarction. In the presence of right bundle branch block, myocardial ischemia/infarction generally still results in the usual ECG changes in leads other than V_1 and V_2, and Q waves in V_1 and V_2 retain their diagnostic significance. For example, **Figure 11.13A** shows the typical rSR' with secondary T wave inversions seen in the septal leads (V_1 and V_2) with right bundle branch block (RBBB). Figure 11.13B shows the inferior leads (II, III, and aVF) from the same ECG. The QS patterns shown are indicative of an old STEMI (Q wave infarct), even in the presence of an RBBB. Other infarct patterns such as ST depression or elevation in leads other than V_1 or V_2 would also retain their usual diagnostic significance in the presence of RBBB. When an infarct involves the septum (the area associated with V_1 and V_2) the situation is slightly more involved. **Figure 11.14** shows cardiograms from two patients with RBBB and infarcts that involved the septum. In Figure 11.14A the ST segment elevations of a STEMI are more apparent, particularly in lead V_2. In Figure 11.14B (from a different patient) the ST elevations are more subtle but still apparent. Notice in both cases

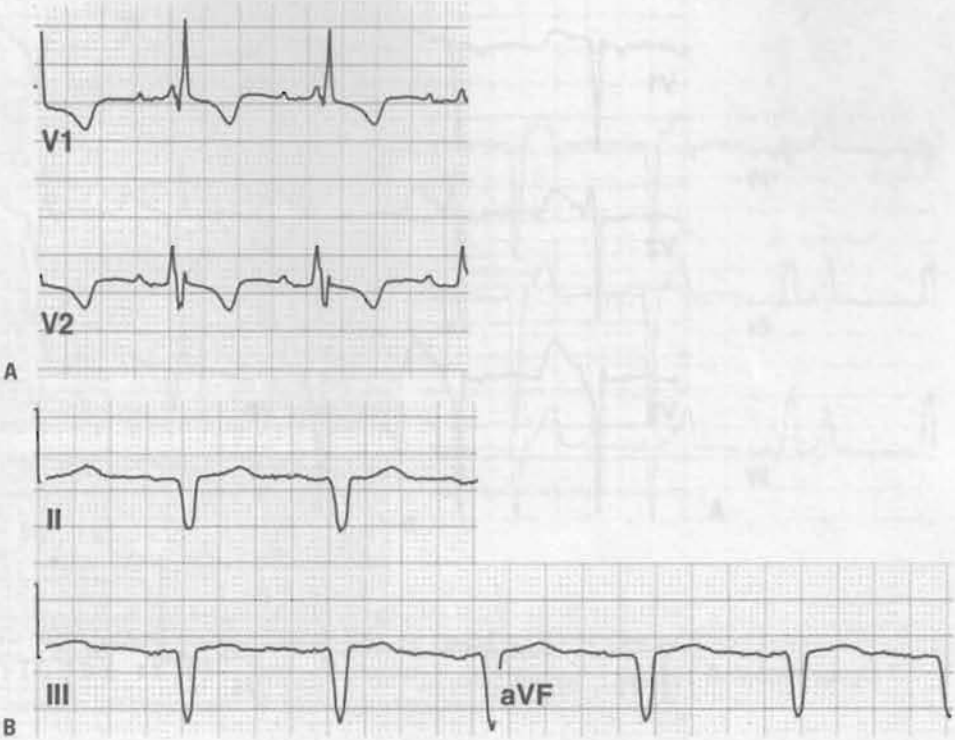


FIGURE 11.13 Old inferior wall infarction with right bundle branch block. **A:** Septal leads. **B:** Inferior leads.

that the T waves are upright, not inverted. This might appear to be normal, but in fact is caused by the infarct. Keeping in mind that T waves in the septal leads should be inverted in the presence of an RBBB (secondary ST-T changes), the upright T waves seen here are actually abnormal and represent primary ST-T changes from the septal infarct. Essentially, upright T waves in these leads in the presence of a bundle branch block are in many ways equivalent to T wave inversions in the absence of a bundle branch block.

A left bundle branch block (LBBB) also complicates diagnosis of an infarct. A new or presumably new LBBB in and of itself greatly raises the index of suspicion for an infarct. This is because infarctions often damage the conduction system and lead to LBBB. With a preexisting LBBB secondary ST-T changes should be present, so similar to the situation with RBBB, primary ST-T changes lead to upright or nearly upright T waves. Notice in **Figure 11.15** that the T waves in the lateral leads are not inverted, although they should be from the LBBB. This is because a lateral wall infarct is also occurring. The infarct causes them to become upright. The changes are present in both panels, although more subtle in Figure 11.15A. The Q waves and ST segment elevations in the right chest leads with LBBB are another complicating factor. These changes make it difficult or impossible to diagnose a septal infarct in the presence of LBBB.

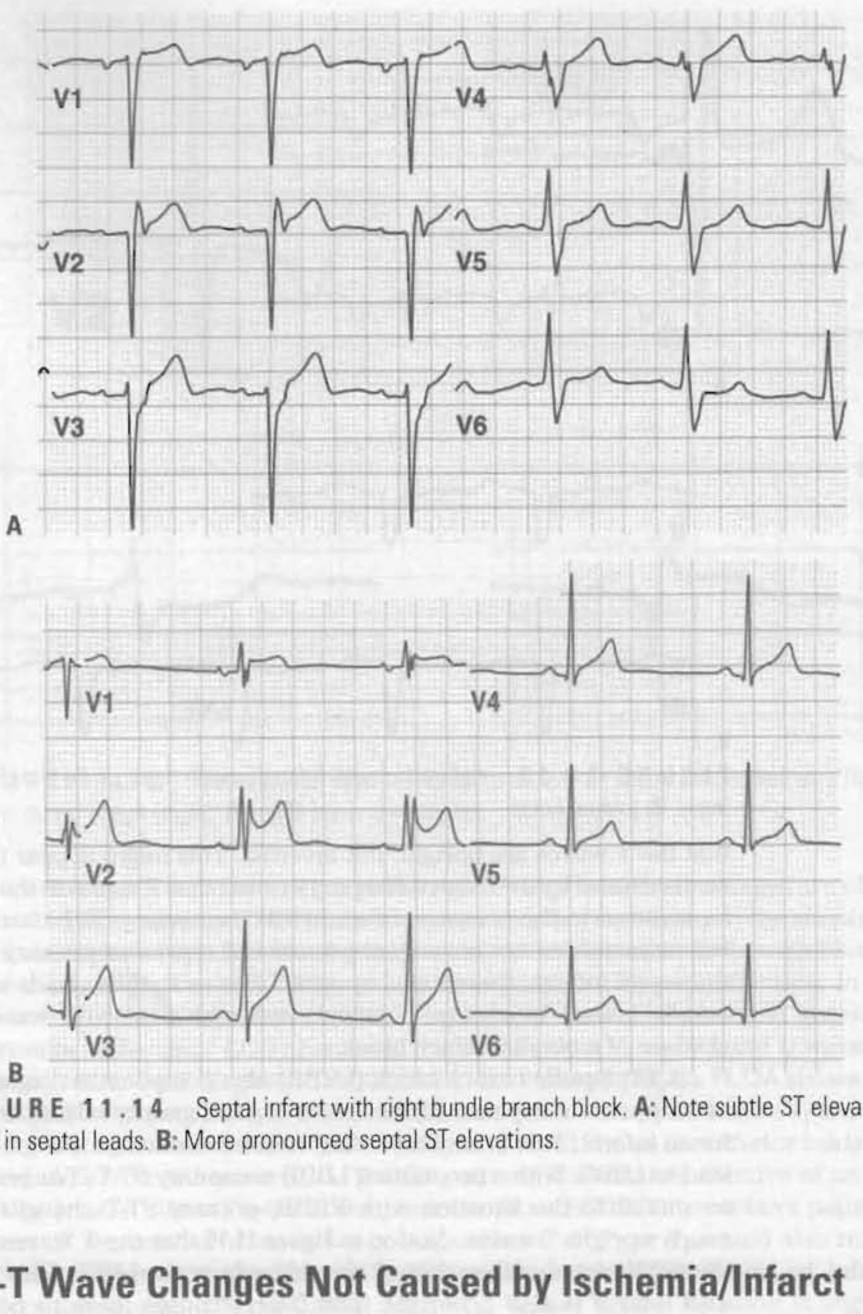


FIGURE 11.14 Septal infarct with right bundle branch block. **A:** Note subtle ST elevations in septal leads. **B:** More pronounced septal ST elevations.

ST-T Wave Changes Not Caused by Ischemia/Infarct

The ST-T wave changes secondary to bundle branch blocks have been described previously (Chapter 10). Changes associated with ischemia or infarction in the presence of conduction defects are discussed above. Hypertrophy patterns are also associated with ST-T wave changes (Chapter 9). For example, the tall R waves seen

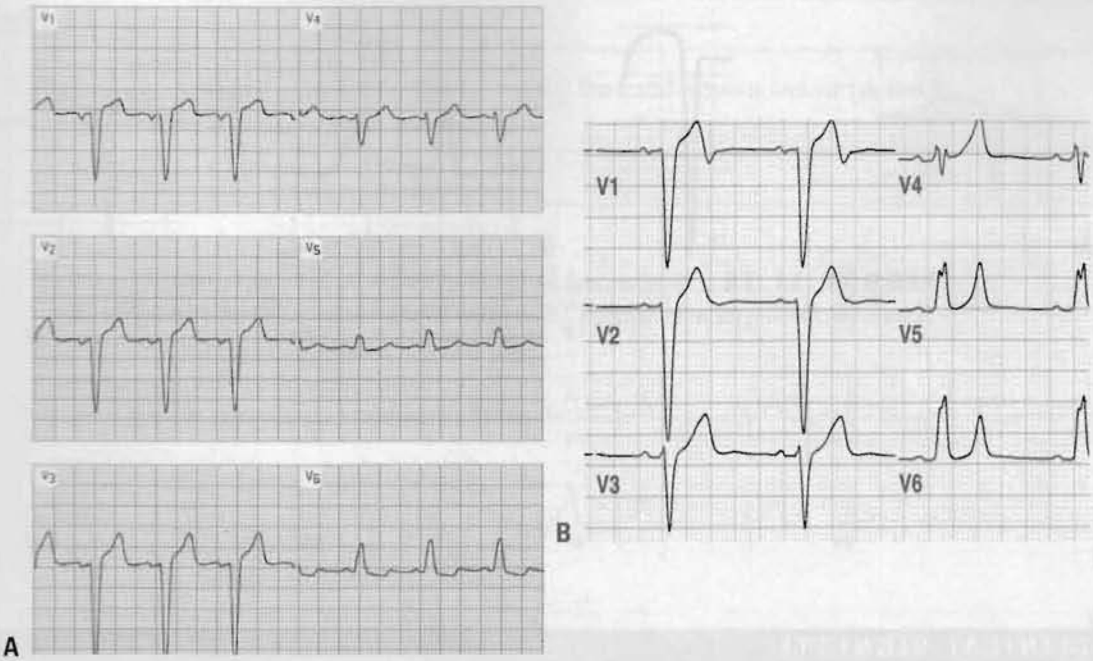


FIGURE 11.15 Left bundle branch block primary ST-T changes. **A:** Modest upright T waves in left chest leads. **B:** More robust positive T waves in V_5 and V_6.

in the lateral precordial leads and/or leads I and aVL with left ventricular hypertrophy may be accompanied by ST segment depression and T wave inversion in a so-called strain pattern. It can be extremely challenging or impossible to accurately diagnose ischemic changes in leads showing a strain pattern as the ST depressions and T wave inversions may be secondary to the hypertrophy or may be caused by ischemia (primary changes). In leads not exhibiting the tall R waves associated with hypertrophy, ST segment depressions and T wave inversions retain their usual diagnostic significance as regards ischemia and/or NSTEMI. In leads that previously showed a strain pattern, the return to an upright configuration (pseudonormalization) for previously inverted T waves is suggestive of ischemia. This is analogous to the reversion to an upright pattern for T waves previously inverted in conjunction with bundle branch blocks. ST segment elevations retain their usual diagnostic significance in the presence of hypertrophy patterns.

Not all ST segment elevations are pathologic. For example, an apparently benign normal variant pattern known as early repolarization (Chapter 12) causes ST segment elevation. A somewhat simplistic but useful observation is that ST segment elevations associated with occlusion of coronary arteries tend to have a somewhat convex appearance said to resemble a "frown" **(Fig. 11.16A)**, while the normal variant type tend to be slightly concave and are likened to a "smile" (Fig. 11.16B).

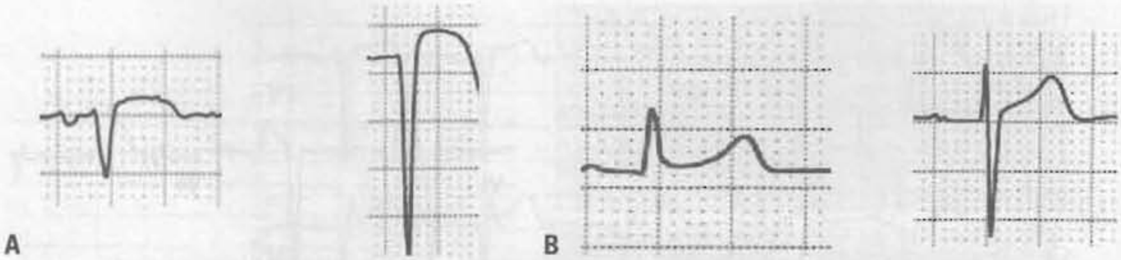
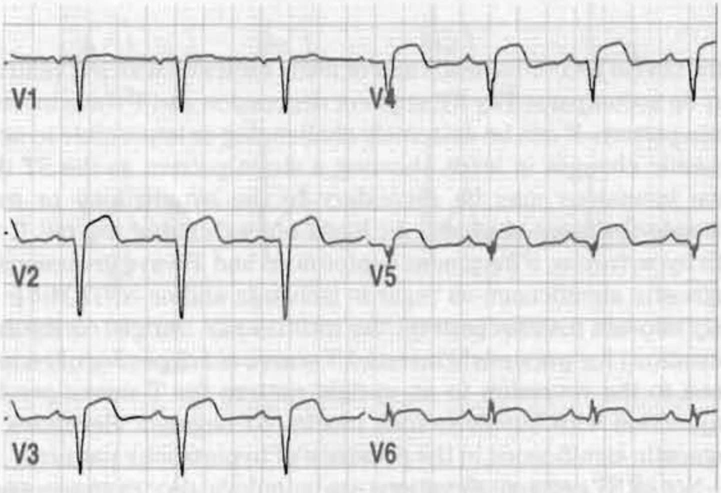


FIGURE 11.16 Ischemic and benign ST elevation. **A:** Ischemic (convex). Looks like a "frown." **B:** Normal variant (concave). Looks like a "smile."

CLINICAL VIGNETTE REVISITED

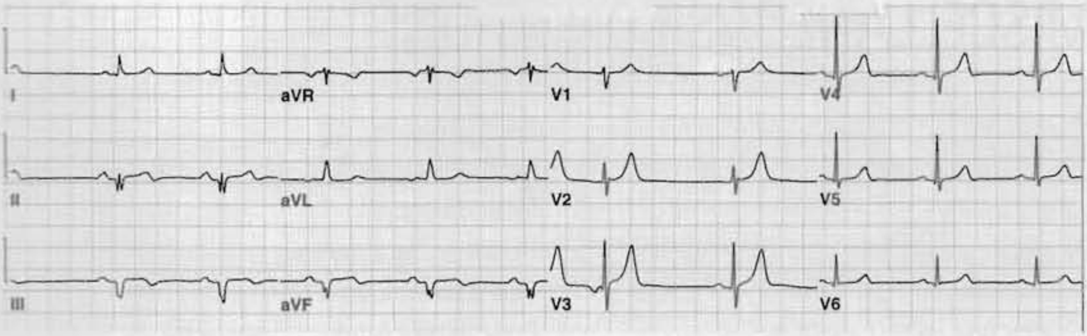
A patient awaiting a stress test complains that he feels a strong pressure in his chest. He appears pale and is sweating profusely.



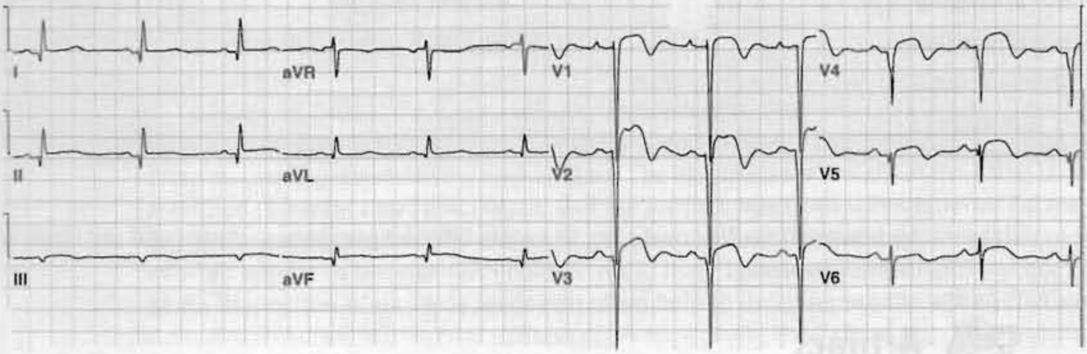
Based on the ECG and symptoms, this patient appears to be experiencing an acute STEMI. After consultation with the cardiologist supervising the stress laboratory, the patient was transported by wheelchair to the emergency department.

Quiz 11

1. Describe any evidence of ischemia/infarction for the cardiograms below (A and B).


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6

A


I
aVR
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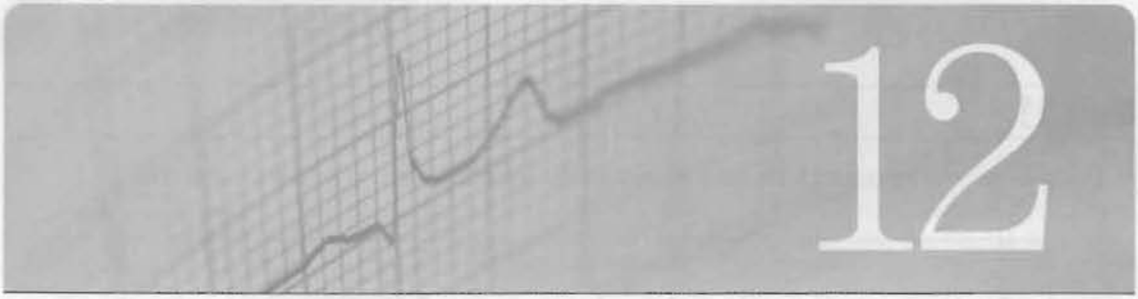
aVF
V3
V6

B



12

MISCELLANEOUS CONDITIONS

- Artifact
- Digitalis Effect
- Pericarditis
- Early Repolarization
- Low Voltage
- Hypo- and Hypercalcemia
- Hyperkalemia
- Hypothermia
- Nonspecific ST-T Wave Abnormalities (Changes)
- Pre-Excitation
- Wolff-Parkinson-White
- Situs Inversus
- Pediatric Cardiograms
- Pulmonary Pattern
- Prinzmetal's Angina (Vasospastic or Variant Angina)
- Pulseless Electrical Activity

A variety of conditions do not fit neatly into the previous sections and are dealt with in this chapter.

KSA Artifact

Patient movement, failure to properly prepare the skin, and use of ECG electrodes that have been stored improperly or are past the expiration date are some common causes of ECG artifact. *Artifact* is particularly common when a patient is exercising. Modern ECG machines typically offer "filters" to reduce artifact by various mechanisms, but artifact still sometimes renders a tracing difficult or impossible to interpret properly.

The various ECG leads often are not uniformly distorted by artifact. **Figure 12.1A** shows an erratic baseline caused by an electrode that was improperly stored and consequently suffered drying of its conductive gel. The electrode in question was placed in the left leg position and therefore caused artifact in leads II and III. Other leads (not shown) were unaffected as the left leg electrode is not involved in their generation (lead I, V_1, etc.). Recalling which electrodes are active for the various leads can be useful as in this example, rather than reprepping and replacing all of the electrodes a simple replacement of the left leg electrode solved the problem.

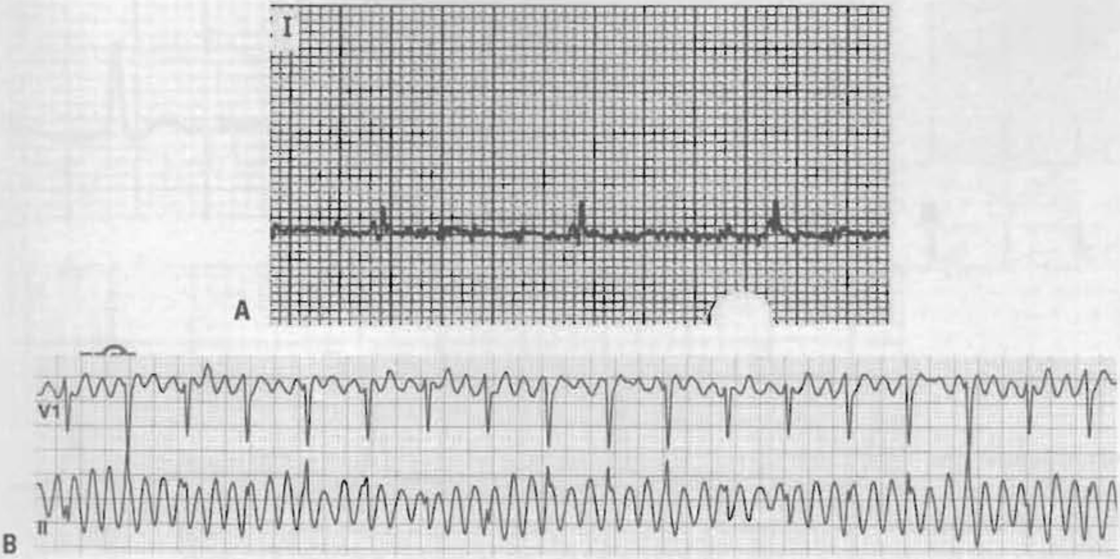


FIGURE 12.1 **A:** Artifact from dry electrode. **B:** Artifact simulating ventricular tachycardia in lead II.

Figure 12.1B shows a patient's ECG viewed simultaneously in leads V_1 and II. Note that in lead II the rhythm appears to be ventricular tachycardia. This strip is from an obese patient during ambulation. The electrodes were all on the torso in the modified (Mason-Likar) placement, and the artifact was from subcutaneous fat moving as the patient walked. By comparing the two leads it becomes apparent that most of what appear to be wide QRS complexes in lead II are actually not QRS complexes at all. Inspection of multiple leads can often help in determining the actual rhythm.

Digitalis Effect

Excessive digitalis (digitalis toxicity) can cause a variety of arrhythmias and types of heart block, but even normal therapeutic doses commonly result in a characteristic "U shaped" (often, as shown, a rather shallow "U") depression of the ST segment **(Fig. 12.2)** known as *digitalis effect*. The presence of digitalis effect does not imply an overdose of the medication, but rather is simply a common finding in patients taking this medication. This complicates ECG interpretation because ST segment depressions due to digitalis can be difficult or impossible to distinguish from ST changes caused by ischemia.

Pericarditis

Pericarditis, an inflammation of the pericardium, results in ST segment elevations that may initially appear similar to those of ST segment elevation myocardial

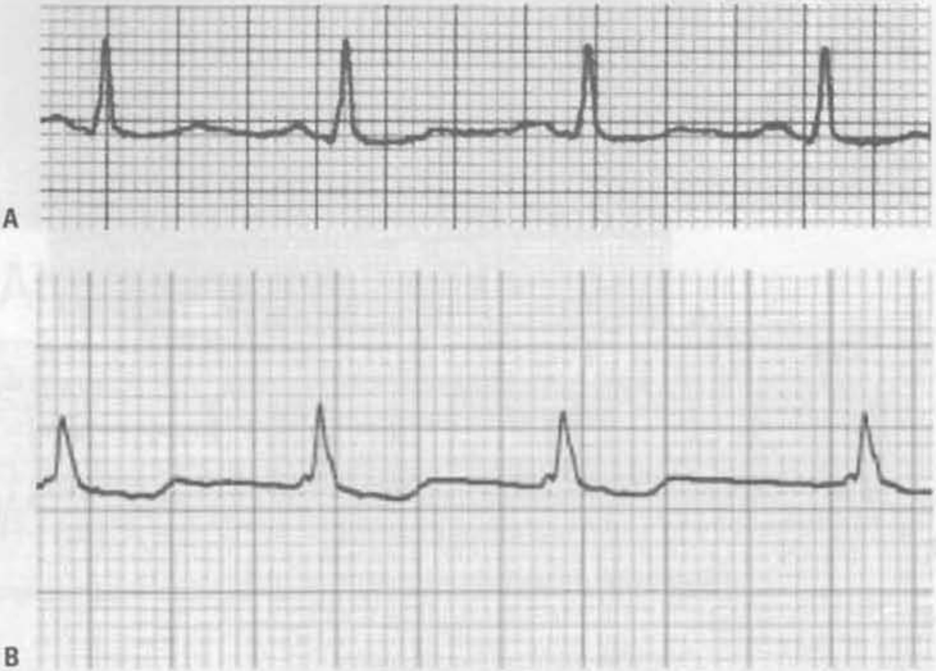


FIGURE 12.2 Digitalis effect. **A** and **B** are two examples of the digitalis effect.

infarction (STEMI). However, the ST elevations of pericarditis differ in several ways from those of an acute infarct.

Pericarditis versus ST Segment Elevation Myocardial Infarction

1. The pericardium surrounds the heart, so pericarditis typically results in ST elevations in all leads except aVR; it is rare for an infarct to cause ST elevations in leads associated with more than one or two regions.
2. The ST segment elevations associated with STEMI change rapidly ("evolve"), as described in Chapter 11, with Q waves commonly developing. The ST elevations from pericarditis are more persistent. The evolution of pericarditis is much slower and involves changes in T wave morphology without the development of Q waves.
3. The T waves are not tall and peaked with pericarditis as they are with STEMI.
4. The PR segments in many leads are depressed below baseline with pericarditis; this does not occur during STEMI.
5. In addition to ST elevations during a STEMI it is common to also see ST segment depression in some leads due to remote ischemia (Chapter 11). With pericarditis all leads with the exception of aVR typically show ST elevations.

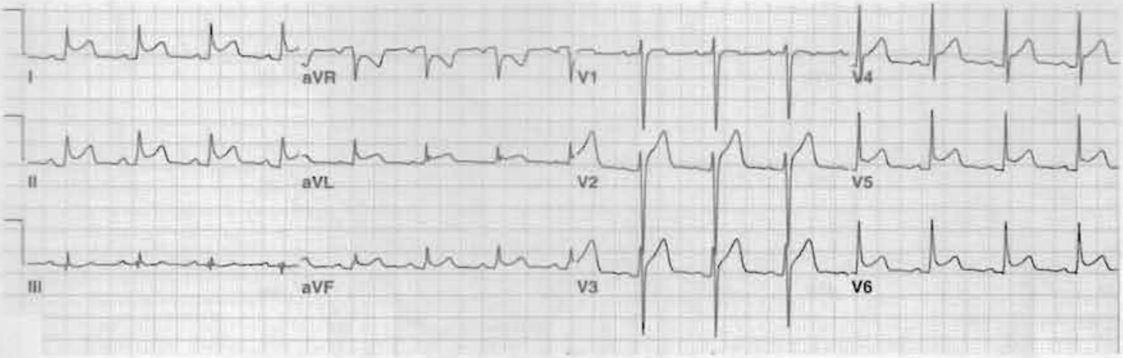


FIGURE 12.3 Pericarditis.

Figure 12.3 is from a patient with pericarditis. Notice that the ST segments are elevated significantly in almost all leads (exceptions are III and aVR), and the PR segments in several leads are mildly depressed (using the TP Segment as baseline). These ST changes persisted for several days and did not go through the evolutionary changes typical of STEMI.

KSA Early Repolarization

Elevation of ST segments does not always indicate infarction or pericarditis. Many apparently healthy individuals show significant (>1 mm) ST elevations, which are a normal variant and are thought to be caused by unusually fast repolarization of the ventricles. A characteristic feature of early repolarization is a brief upward deflection in one or more leads at the J point (where the QRS complex ends and the ST segment begins). Although subtle, this upward deflection can be seen clearly at the beginning of the ST segment in lead V_3 in the area circled in **Figure 12.4** and is also visible in the inferior leads and V_5 and V_6. These changes are not typically seen globally as in pericarditis, although they often occur in multiple leads. The ST elevations of early repolarization do not change over time as those of STEMI do.

KSA Low Voltage

In some instances the electrical activity of the heart is less than normal. For instance, this may occur due to hypothyroidism or following a large infarct that results in the death of numerous myocardial cells. In other cases the heart's electrical activity is within normal limits, but the current recorded by the surface electrodes used in a standard ECG is below normal as a result of resistance to current flow because of factors such as large amounts of subcutaneous fat or increased intrathoracic air volume (e.g., emphysema). Various definitions of low voltage are in clinical use. One definition is a total QRS voltage of ≤5 mm (0.5 mV) in each of the limb leads. In **Figure 12.5** no QRS has a total voltage (including positive and

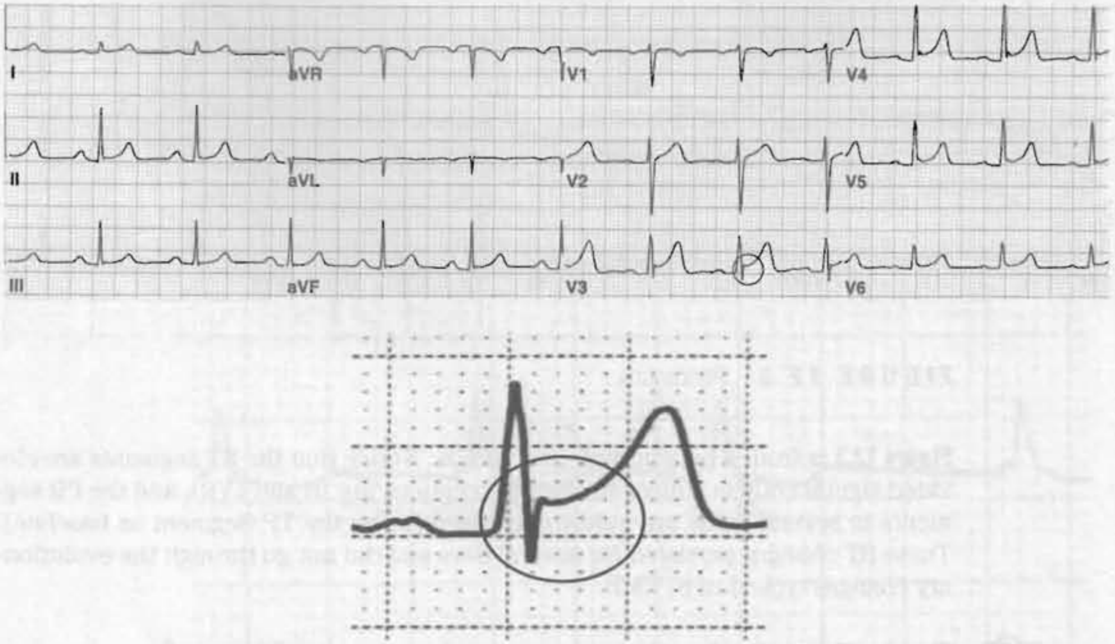


FIGURE 12.4 Early repolarization. Note the small "notch" at the J point (*circled*).

negative deflections) of 5 mm (0.5 mV) or greater in any of the limb leads (I, II, III, aVR, aVL, aVF). Another criterion is no precordial lead having a total voltage (R and S together) of >15 mm (1.5 mV). In the authors' opinion both criteria should be met (total QRS voltage of ≤5 mm in each limb lead and ≤15 mm in each precordial lead). Because these ECG findings may be from diverse causes, a specific diagnosis cannot be made based on the ECG alone, and such tracings may simply be described as low voltage QRS complex.

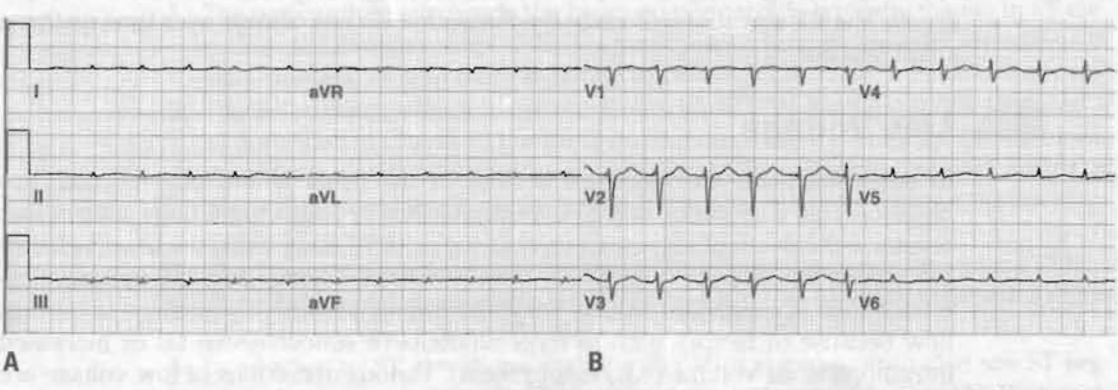


FIGURE 12.5 Low voltage. **A:** Limb leads with no QRS >5 mm. **B:** Precordial leads with no QRS >15 mm.

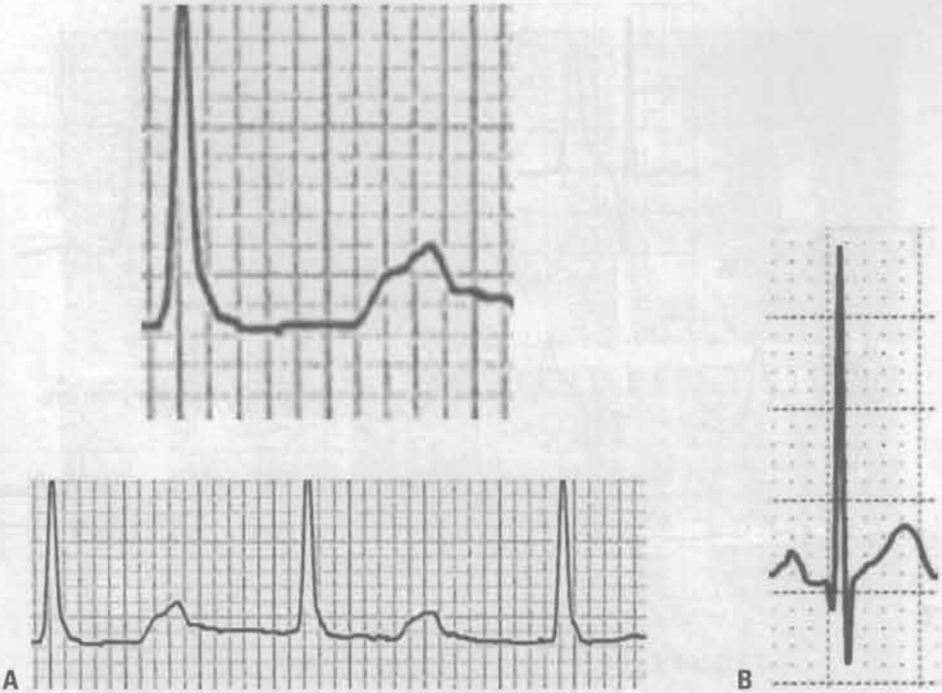


FIGURE 12.6 **A:** Hypocalcemia (long QT). **B:** Hypercalcemia (short QT).

Hypo- and Hypercalcemia

Low (hypo) or high (hyper) plasma calcium levels are associated with specific ECG findings. *Hypercalcemia* decreases the QT interval and *hypocalcemia* prolongs it **(Fig. 12.6)**. It is difficult to provide universally applicable numerical cutoffs for what constitutes a short QT interval; however, when the ST segment is essentially not present (the beginning of the T wave comes right after the QRS complex), it is reasonable to assume that the QT is short with hypercalcemia as a likely cause. Prolongation of the QT interval is determined by the usual methods (Chapter 1), and hypocalcemia is only one possible cause of a prolonged QT interval.

Hyperkalemia

Hyperkalemia, elevated plasma potassium, causes a variety of changes on the cardiogram depending on how elevated the potassium levels are. Arguably the most important change is the appearance of tall, peaked T waves **(Fig. 12.7)**, as this is the initial finding. As serum potassium levels continue to rise the T waves remain tall and peaked and what appears to be an idioventricular rhythm* often ensues

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*Although it appears to be idioventricular, it actually is a sinoventricular rhythm, which is unique to hyperkalemia.

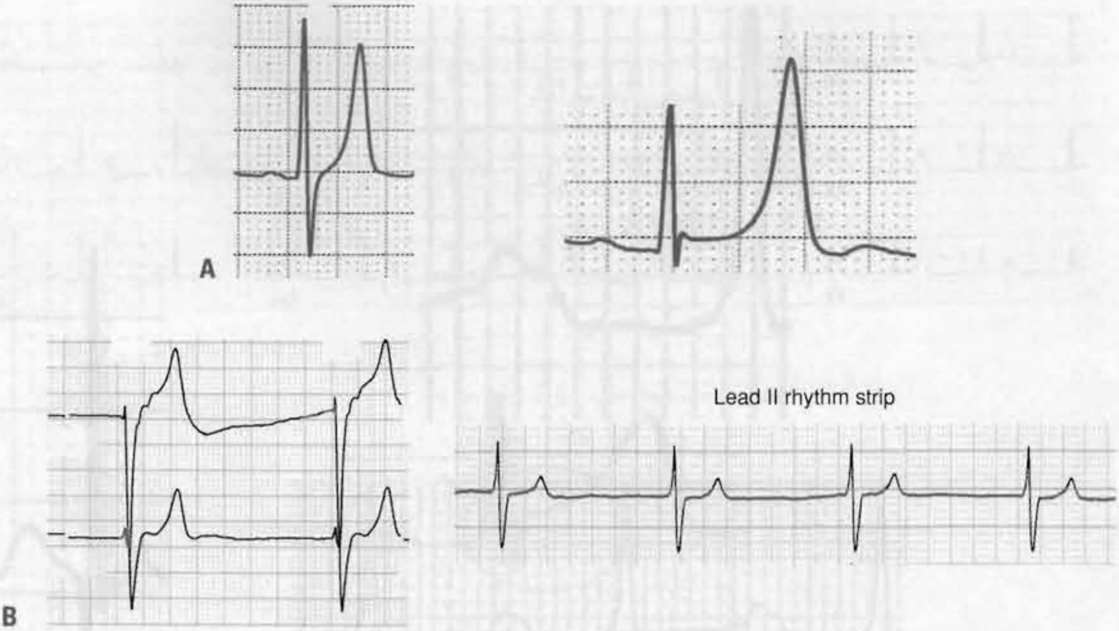


FIGURE 12.7 **A:** Early hyperkalemia. **B:** Progressing hyperkalemia.

(progressing; Fig. 12.7). Patients at this or later stages would be too sick to appear in a stress lab or cardiac rehabilitation facility.

Hypothermia

In some people severe hypothermia can lead to characteristic ECG changes. The ECGs of **Figure 12.8** are from the same patient during hypothermia and following successful rewarming. Notice that during the hypothermic state the J point is elevated and the T wave takes off immediately after the J point. These ST-T wave changes are known as *Osbourne waves*. With achievement of a normal core temperature, the Osbourne waves are no longer present. Not all patients will develop Osbourne waves with hypothermia. Sinus bradycardia and atrial fibrillation are also commonly seen with hypothermia.

Nonspecific ST-T Abnormalities (Changes)

As previously mentioned, specific T wave patterns are associated with specific abnormalities. For instance, deep asymmetrical T wave inversions occurring in conjunction with tall R waves are suggestive of ventricular hypertrophy and represent the strain pattern (or more properly, repolarization abnormality pattern). Many patients exhibit subtle T wave and/or ST segment abnormalities ("changes") that deviate from the norm but are not robust enough to be diagnostic for any particular

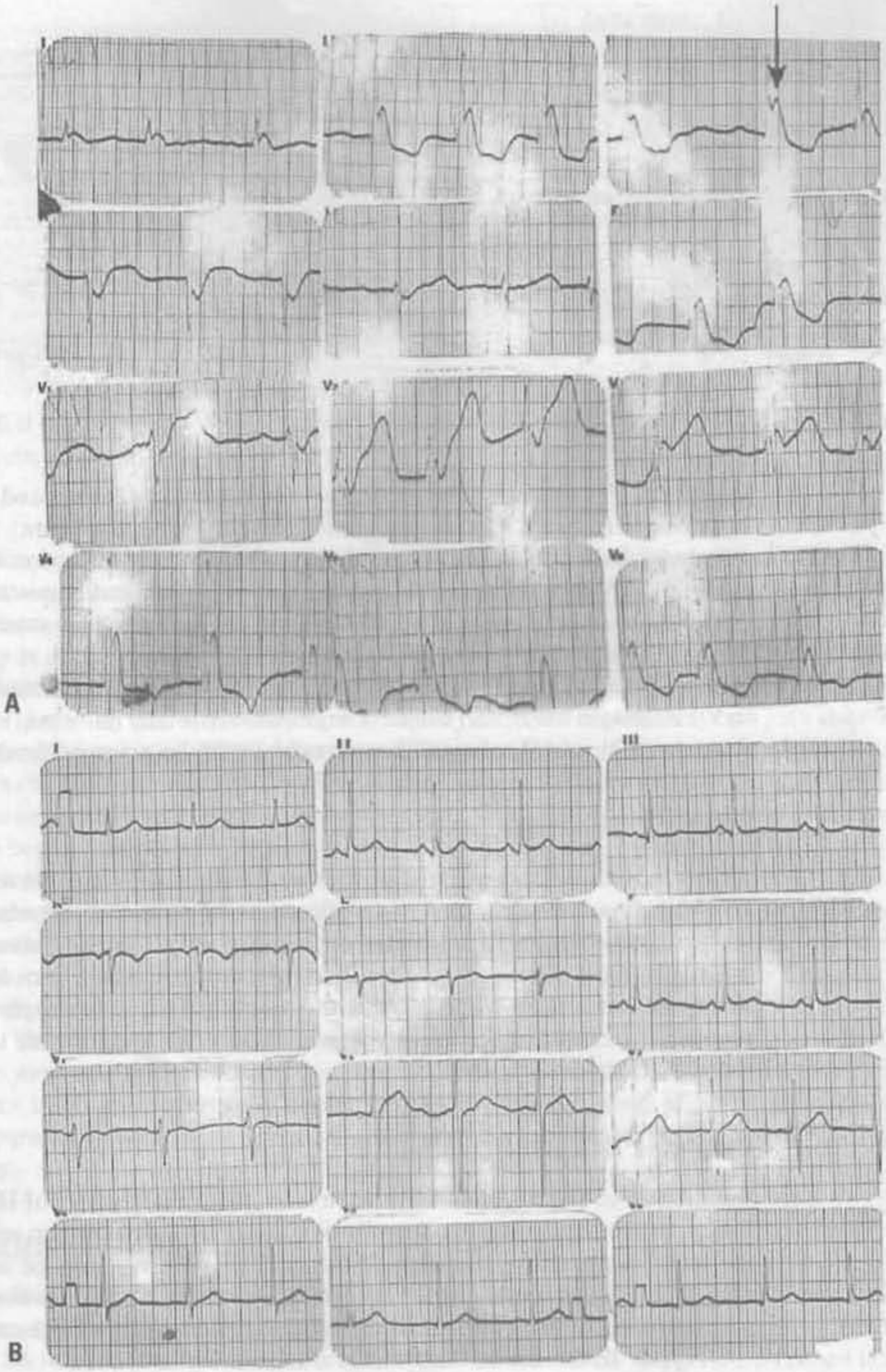

A
I
II
III
V1
V2
V4
V5
V6
B
I
II
III

FIGURE 12.8 Osbourne waves. **A:** Hypothermia. Note the Osbourne waves (*arrow*). **B:** Hypothermia rewarmed.

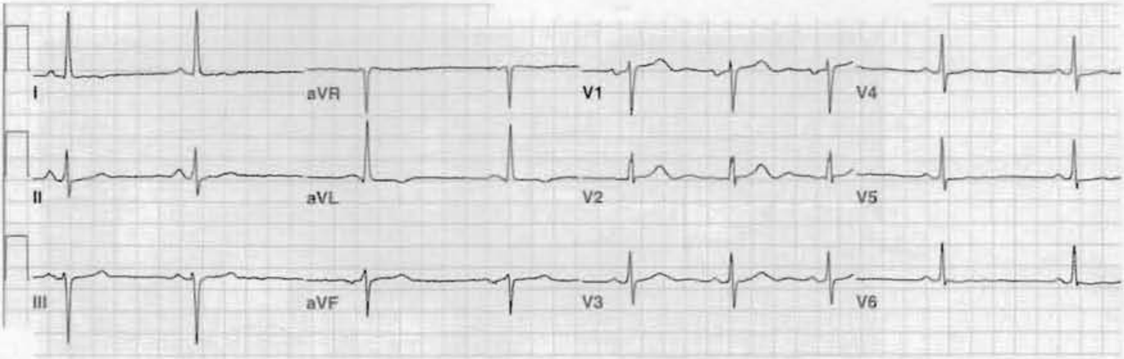


FIGURE 12.9 Nonspecific ST-T wave abnormalities.

condition. For example, the ST segment may be slightly elevated or depressed, but not enough to reach specific diagnostic criteria (typically 1 mm). Similarly T waves may be abnormal in that they are positive but very small, nonexistent or very subtly inverted. None of these situations meets any specific diagnostic criteria, but yet each is a deviation from the norm. Collectively such findings are referred to as nonspecific ST-T abnormalities or nonspecific ST-T changes.

In **Figure 12.9**, the T waves are very small (II, III, aVF) or virtually nonexistent (V_4 to V_6) in certain leads, and some ST segments are mildly elevated (V_1 and V_2) or mildly depressed (V_4 and V_5). Any of these would qualify as nonspecific abnormalities.

Pre-Excitation

Pre-excitation is characterized by an abnormally short delay between the beginning of atrial depolarization and the beginning of ventricular depolarization. In some types of pre-excitation such as Lown-Ganong-Levine (LGL), the only ECG finding is a PR interval <120 ms (0.12 sec). It is not possible to tell from a standard 12-lead ECG whether a short PR interval is the result of LGL or is simply a normal variant, so when interpreting such a cardiogram it may be appropriate to simply indicate that the PR interval is short.

Wolff-Parkinson-White

In addition to the normal atrioventricular (AV) node/bundle of His electrical pathway connecting the atria and ventricles, some patients have an extra atrioventricular "wire" termed an *accessory pathway*. This pathway may be active regularly, in which case ECG evidence will be visible, or it may be a concealed pathway that is not active at all times. When the pathway is concealed the ECG may appear normal.

Figure 12.10 shows the three features (triad) of Wolff-Parkinson-White (WPW). When active, the accessory pathway of WPW allows depolarizations to travel from atria to ventricles via two paths: the normal AV node/bundle of His

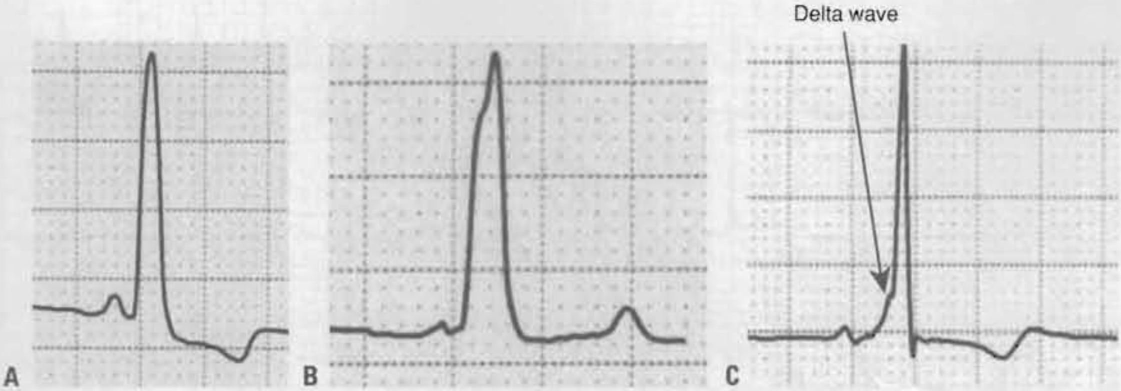


FIGURE 12.10 Wolff-Parkinson-White triad. **A:** Short PR interval. **B:** Wide QRS complex. **C:** Delta wave.

pathway and the accessory pathway (also known as the bundle of Kent). The accessory pathway does not have the "delay" inherent in the AV node, so depolarizations travel rapidly from atria to ventricles. Although the accessory pathway is allowing a more rapid AV conduction, the normal AV nodal/bundle of His depolarizations are also proceeding. These rapid depolarizations by the accessory pathway and the normal (and slightly slower) depolarizations join together at the beginning of ventricular depolarization. Early repolarization of the ventricles via the accessory pathway results in a short PR interval; merging of the early (via accessory pathway) and normal (via AV node) depolarizing currents widens the beginning of the QRS complex in a distinctive way, resulting in the wide QRS with the base of the QRS said to resemble the Greek letter delta (delta wave). **Figure 12.11** shows two 12-lead ECGs from patients with WPW. The previously mentioned triad of a short PR interval, wide QRS complex, and a delta wave can be located in each cardiogram, however, these changes will not be found in all leads.

These accessory pathways are of more than academic concern because they can serve as a conduit for re-entry tachycardias. **Figure 12.12** shows two ECGs from the same patient. In Figure 12.12A the classic triad of WPW can be seen. Figure 12.12B shows a re-entrant tachycardia facilitated by the accessory pathway.

Situs Inversus

Occasionally an ECG similar to that shown in **Figure 12.13** is encountered. The P waves are negative in lead II and positive in aVR, the axis is extremely deviated (in fact the opposite from the norm), and the r waves are tiny in the leftward leads (I, aVL, V_5, V_6). In many respects this cardiogram exhibits findings opposite from what would normally be expected. It would be appropriate to check that the

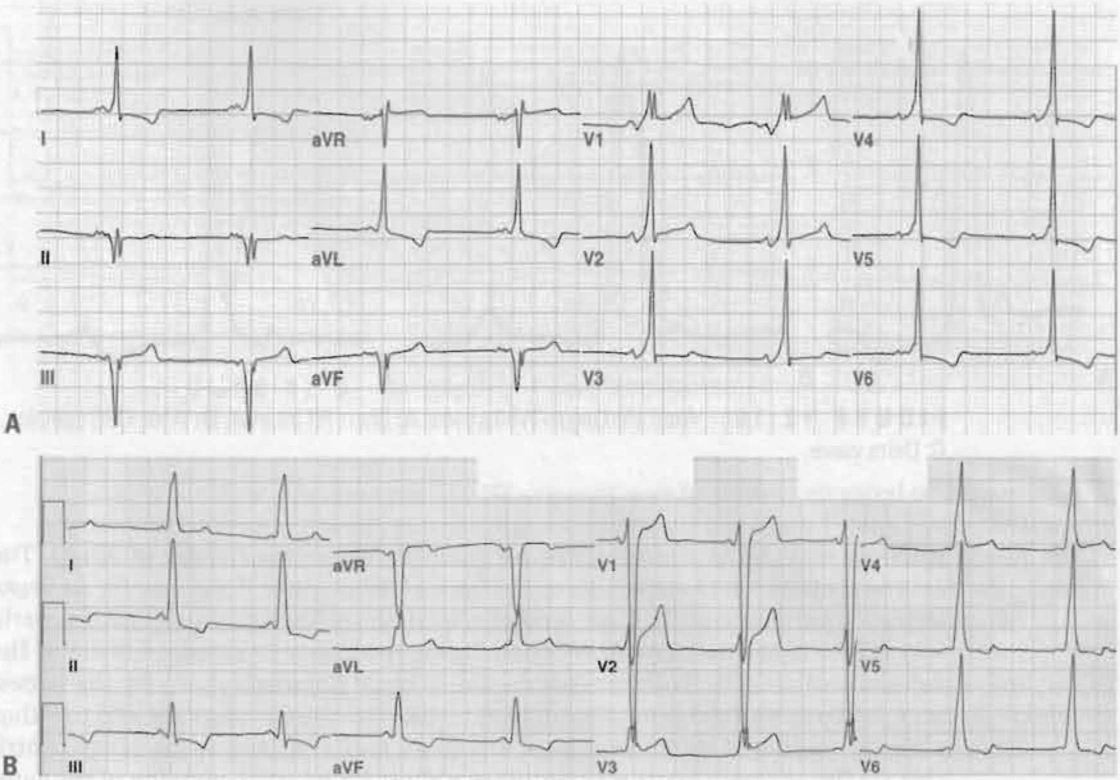


FIGURE 12.11 Wolff-Parkinson-White. **A** and **B** are two examples of Wolff-Parkinson-White.

wires are attached to the proper electrodes or establish by other means (voltage in I + III = II, etc.) that these findings are not caused by technical error. In this example the cause of this "backward" cardiogram is not technical error, but rather a reversal of the position and orientation of the viscera known as *situs inversus*. The heart and other major organs are reversed relative to the norm, therefore, the electrical activity of the heart is largely the opposite of the norm. Reversing the placement of the arm and leg leads (RA placed on LA, etc.) and placing the precordial leads on the right side of the chest (as described in Chapter 11 for right-sided infarcts) will allow appropriate recording of an ECG for these patients.

Pediatric Cardiograms

This text is concerned with interpretation of adult ECGs. The normal and abnormal ECG patterns of pediatric patients differ in important ways from adults. Even a brief inspection of the normal pediatric (neonatal) ECG shown in **Figure 12.14** should illustrate this concept. Note for example, the fast rate, the relatively tall R

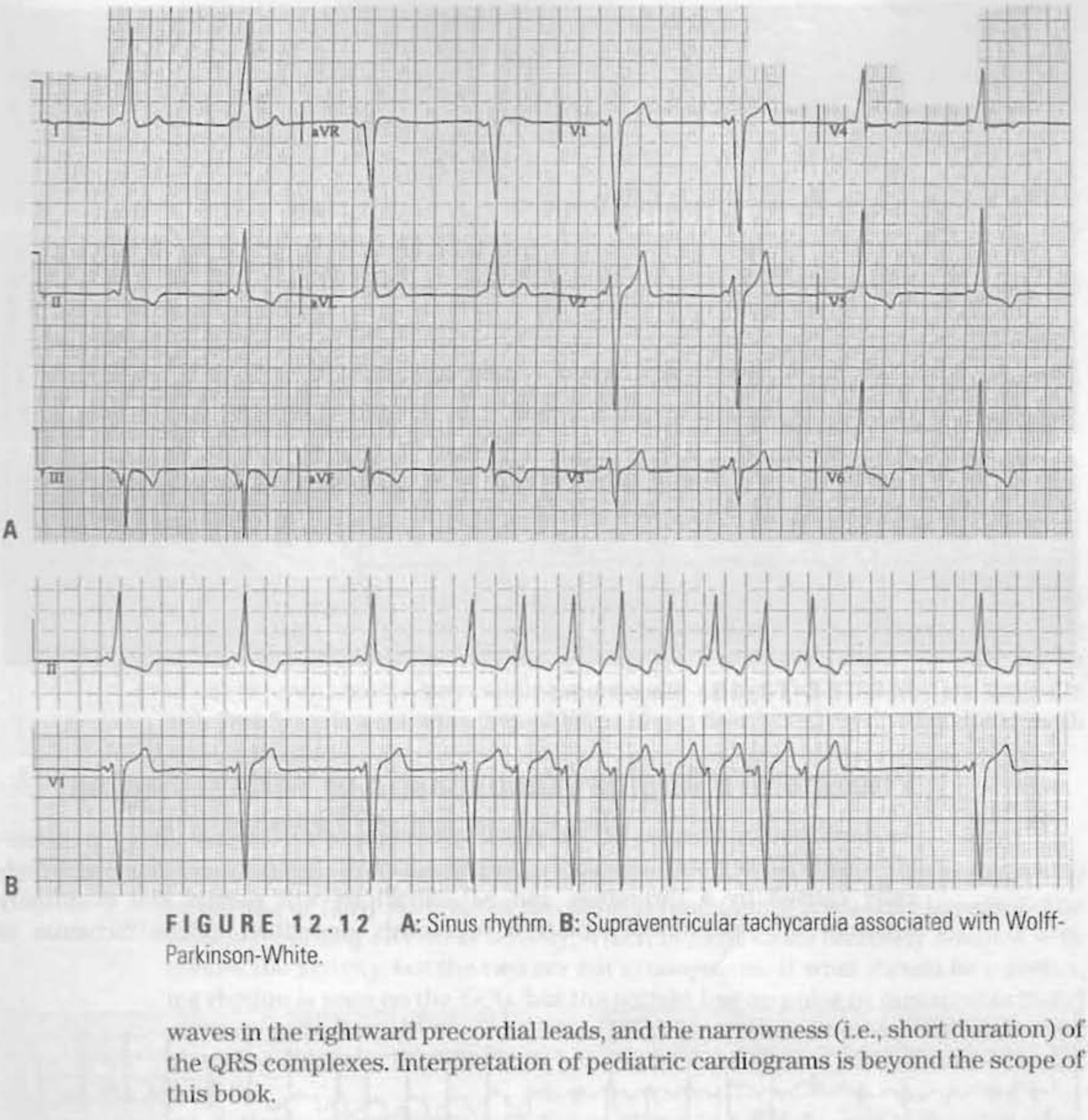


FIGURE 12.12 **A:** Sinus rhythm. **B:** Supraventricular tachycardia associated with Wolff-Parkinson-White.

waves in the rightward precordial leads, and the narrowness (i.e., short duration) of the QRS complexes. Interpretation of pediatric cardiograms is beyond the scope of this book.

Pulmonary Pattern

Acute overload of the right heart can result in a distinctive ECG pattern variously referred to as a pulmonary pattern, cor pulmonale, or right heart strain **(Fig. 12.15)**. The hallmarks of the pattern are an S wave in lead I and Q waves and T wave inversions (usually shallow) in lead III. This combination is commonly referred to in shorthand as "S1-Q3-T3." Various lung disorders, including pulmonary embolism, can cause an S1-Q3-T3.

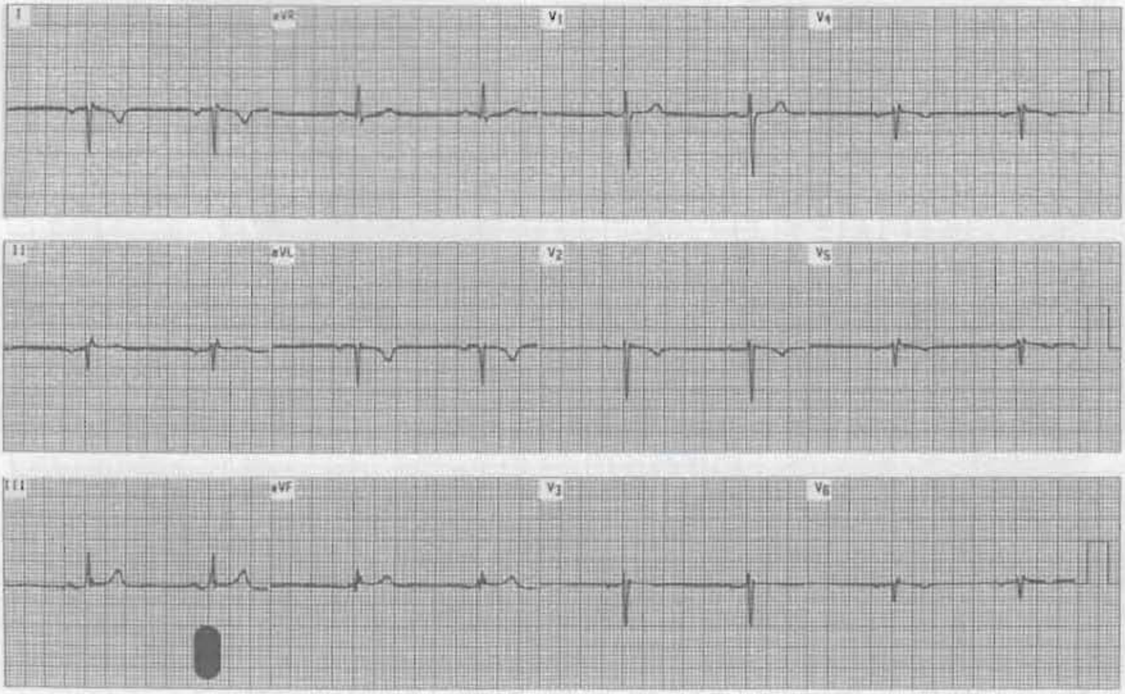

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6

FIGURE 12.13 Sinus inversus.

Prinzmetal's Angina (Vasospastic or Variant Angina)

As described in Chapter 11, complete occlusion of a coronary artery is associated with ST segment elevation. During a STEMI, arterial occlusions are typically caused by a thrombus, and ST elevations will persist and eventually undergo the evolutionary pattern preciously described unless ischemia is

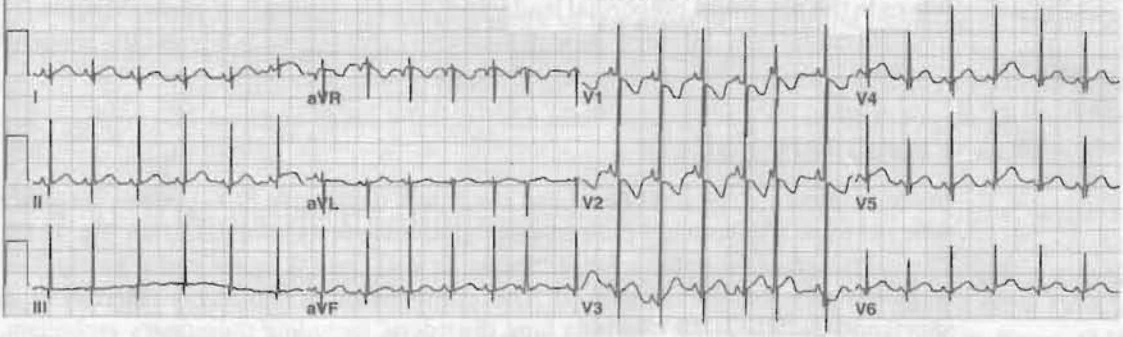

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6

FIGURE 12.14 Pediatric ECG.

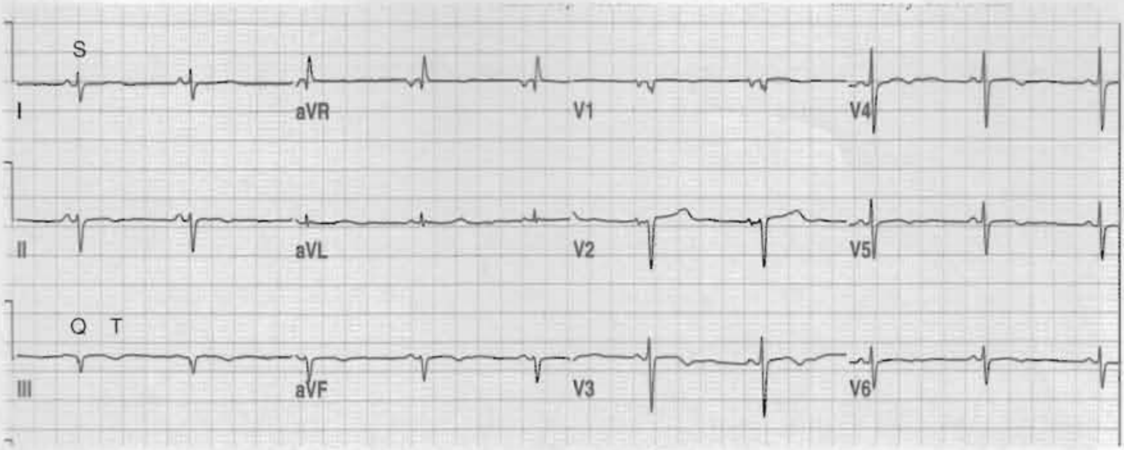


FIGURE 12.15 S1-Q3-T3 (pulmonary pattern).

relieved by interventions such as thrombolytic therapy or angioplasty. In contrast, a condition variously known as Prinzmetal's angina, vasospastic angina, or variant angina results in transient ST segment elevations caused by contraction of the smooth muscle surrounding a coronary artery. The ST elevations typically only last for a few minutes **(Fig. 12.16)** as blood flow is restored when the smooth muscle relaxes.

Pulseless Electrical Activity

In some settings (e.g., cardiac tamponade), the electrical activity of the heart may be normal or fairly normal but mechanical functioning is severely impaired. The ECG is recording electrical activity, which in most cases is closely coupled with contractile activity, but the two are not synonymous. If what should be a perfusing rhythm is seen on the ECG, but the patient has no pulse or measurable blood pressure pulseless electrical activity (PEA) is present. As such, PEA cannot be identified solely by ECG findings, but requires correlation with clinical observations.

The term electromechanical dissociation was formerly used to describe situations where the electrical activity of the heart (ECG) was uncoupled from the mechanical activity. The newer term pulseless electrical activity is now preferred, but both terms are instructive as the electrical activity of the heart is dissociated from the mechanical, and due to the low or nonexistent cardiac output, no pulse is present. The ECG may show virtually any rhythm, including normal sinus rhythm, but no pulse or measurable blood pressure is present. Without intervention the lack of meaningful cardiac output will quickly lead to severe clinical deterioration and death.

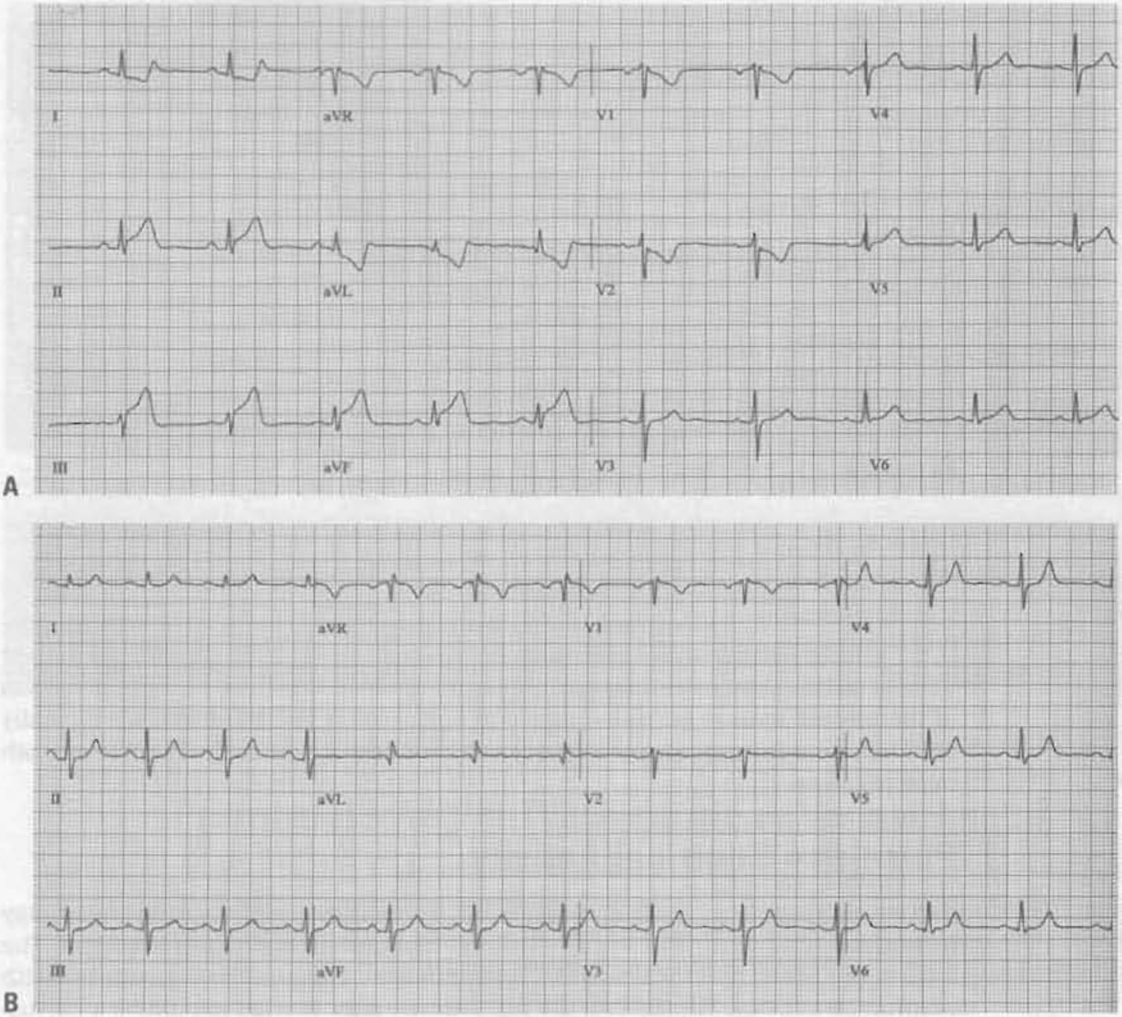

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
A
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
B

FIGURE 12.16 Prinzmetal's angina. **A:** First ECG. **B:** Four minutes later.

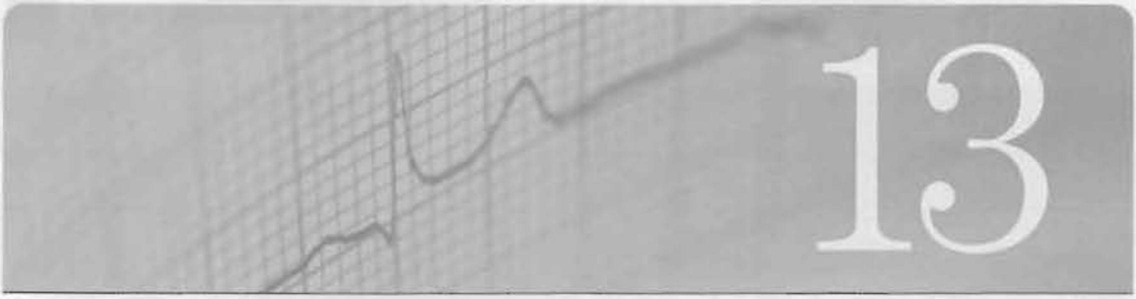
Quiz 12

Some conditions described in this chapter result in ECG patterns similar to those associated with abnormalities described in previous chapters. For each set of conditions below compare and contrast the different ECG patterns.

1. Pericarditis versus STEMI.

2. Early repolarization versus STEMI.

3. WPW versus conduction defects.



13

SYSTEMATIC INTERPRETATION OF ECG AND REVIEW

Although some new concepts are introduced, this chapter largely reviews what has already been introduced and provides a framework for systematic evaluation of ECGs. A variety of parameters (PR interval, QRS duration, relationship of P waves to QRS complexes, etc.) must be evaluated for proper ECG interpretation. The task can seem overwhelming and mistakes will be made if a systematic strategy is not used. The particular system used is probably not as important as is the use of an approach that is organized and inclusive. This chapter will describe a logical system that, after determination of rate, begins with examination of the P waves and proceeds to the right. **Table 13.1** summarizes the steps. The capitalized words highlight which areas of interpretation are covered during each step. We will return to this system following a brief review of T wave patterns.

T Waves

In previous chapters a variety of T wave patterns were encountered, and they are summarized in **Figure 13.1**. Very small (positive or negative) or virtually absent T waves fall into the "nonspecific T wave abnormality" category. Deep, symmetrically inverted T waves represent either ischemia or the repolarization abnormalities ("strain") associated with hypertrophy. In order to be considered indicative of strain they must appear in leads showing R waves meeting voltage criteria for hypertrophy. As ischemia commonly occurs in hypertrophied hearts it is still not possible to rule out ischemia in such cases.

Deep or shallow T wave inversions can also occur with an evolving ST segment elevation myocardial infarction (STEMI). The ST segments may still be elevated (although not as much as during the acute phase) or may have returned to baseline.

Tall, peaked T waves occur with early hyperkalemia or very early in the course of a STEMI.

TABLE 13.1

Systematic Examination of ECG

Rate	What is the rate? For many purposes it is sufficient, at least initially, to simply establish if it is slow, fast or normal. RHYTHM Is the rate (rhythm) regular? RHYTHM
P waves	Are they positive in lead II? RHYTHM Are they unusually tall or wide in lead II? HYPERTROPHY Is the negative part of the P in lead V_1 1 mm wide and deep? HYPERTROPHY Is the PR interval normal and consistent? RHYTHM, AV BLOCKS Is one P present for each QRS complex? RHYTHM, AV BLOCKS
QRS complexes	Is the QT interval normal? QT PROLONGATION, DRUG EFFECTS, ELECTROLYTE DISTURBANCES Are abnormal Q waves present? INFARCT, CONDUCTION DEFECTS Is the QRS wide in any of the limb leads? RHYTHM, CONDUCTION DEFECTS What is the axis (for many purposes it is not necessary to determine degrees)? AXIS, CONDUCTION DEFECTS Are any of the criteria for LVH met? HYPERTROPHY Is the height of the R waves in $V_1 \geq$ the depth of the S waves? HYPERTROPHY, INFARCT LOW VOLTAGE?
ST segments and T waves	Are the ST segments elevated or depressed? ISCHEMIA/INFARCTION, CONDUCTION DEFECTS, HYPERTROPHY Are the T waves normal in appearance and orientation? ISCHEMIA/INFARCTION, CONDUCTION DEFECTS, HYPERTROPHY, ELECTROLYTE DISTURBANCES

Systematic Interpretation of ECGs: the P QRS-T Method

It is important to perform all of the steps shown in Table 13.1, but temporary deviation from the pattern is often useful for exploring particular issues. For example, the discovery of negative P waves in lead II should lead to a search for the cause (technical problem, rhythm disturbance, etc.). Once an explanation is found return to the P wave step and continue from there. An advantage of this system is that a list of questions to answer need not be memorized. As one moves systematically forward from P waves to T waves all of the pertinent issues (rhythm, hypertrophy, ischemia/infarct, etc.) will be addressed.

Use the method described above (or any other comprehensive and systematic method) to interpret the cardiograms that follow. Most of these ECGs are complex and would challenge the average physician. They are intentionally complicated in order to review as much as possible and to help instill an appreciation of the value of a systematic approach to ECG analysis. Most ECGs are not as difficult as these examples; in many areas of clinical practice, it is common to encounter cardiograms

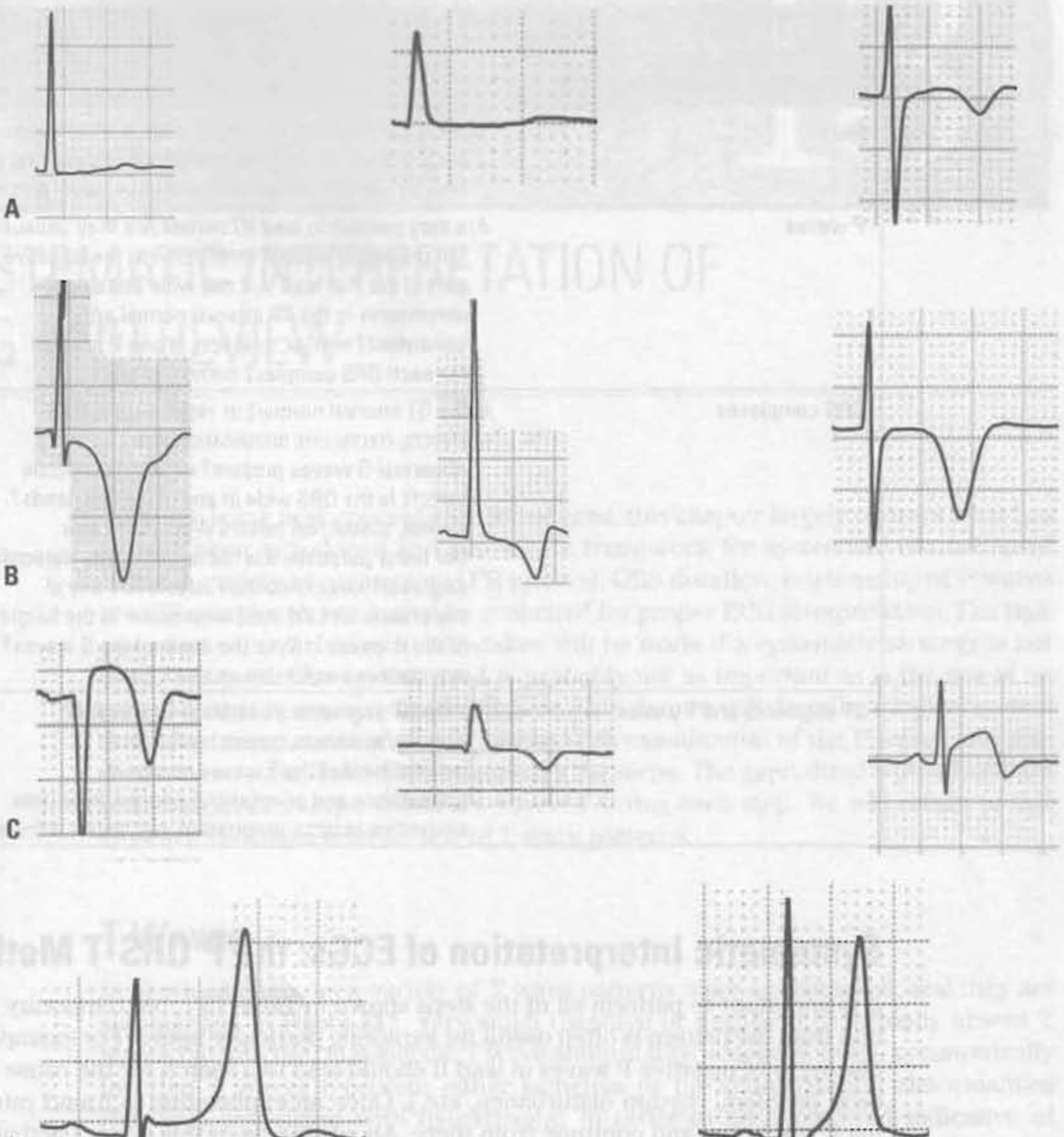


FIGURE 13.1 T waves. **A:** Nonspecific. **B:** Ischemia or "strain" (hypertrophy). **C:** Evolving infarct. **D:** Hyperkalemia or very early infarct.

devoid of abnormalities that may simply be described as *normal sinus rhythm, normal QRS complex configuration, normal ST segments and T waves*.

Do not be discouraged if the exact cause of all of the abnormalities is not readily apparent. Particularly when first starting out one of the main skills is to recognize that something is abnormal and seek appropriate guidance. For example, if while working in a stress lab you encounter a resting ECG that you recognize as abnormal but are not sure what the problem is it is usually appropriate to seek guidance before proceeding with the test. As you encounter various abnormalities in the field and discuss cardiograms with colleagues your ECG knowledge will grow.

Figure 13.2 shows a normal sinus rhythm with complete heart block and an idioventricular escape rhythm, left axis deviation.

Rate: The atrial rate is 91 and regular, the ventricular rate is 26 beats per minute (bpm) and regular.

P: The P waves are upright and of normal appearance. Many of the P waves are not followed by QRS complexes, those that are have differing PR intervals (the P-P intervals are consistent). The atrial rates and ventricular rates are regular and seem to have no relationship to each other; the atrial rate is faster than the ventricular rate. The rhythm is complete (third degree) atrioventricular block.

QRS-T: The QRS complexes are wide and bizarre in appearance and have a left bundle branch block morphology (QS in V_1, wide R in V_6). Given that the rhythm is

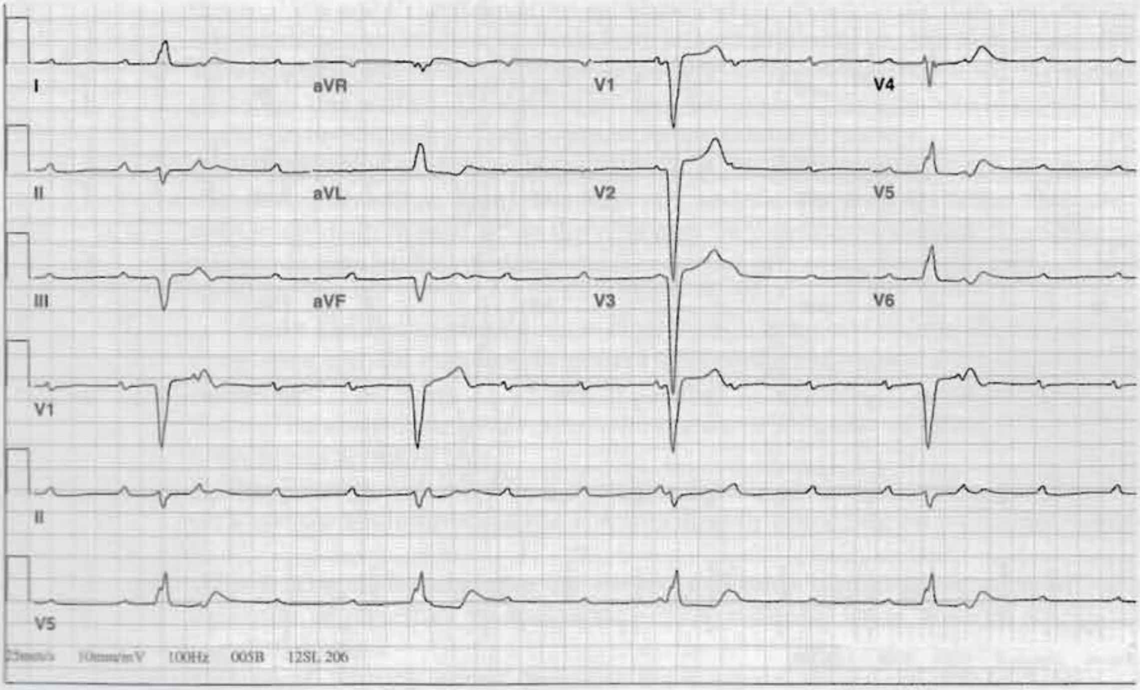


FIGURE 13.2

complete heart block, this likely represents an idioventricular escape rhythm. If one P wave preceded each QRS complex in a normal fashion, then the same QRS morphology would represent left bundle branch block. As the appearance of the QRS is explained by the idioventricular rhythm, this is not a conduction defect, although some clinicians would add the term "with a left bundle branch block morphology" to the interpretation to infer where in the ventricles the idioventricular rhythm is originating. The QS complexes or small r waves and ST segment elevations in the rightward precordial leads are explained by the abnormal conduction of the ventricular beats (analogous to what occurs with left bundle branch block); the QS complexes in leads III and aVF are not due to an old inferior wall infarct (although it would be reasonable for the beginning/intermediate student of ECG to assume that they are) but are associated with the abnormal conduction of the idioventricular rhythm. The axis is left axis deviation (approximately −60); since the altered mean QRS vector is explained by the ventricular origin of the impulses, this does not imply a left anterior hemiblock.

Figure 13.3 shows normal sinus rhythm with frequent premature atrial complexes (PACs) and a ventricular couplet, old inferior wall infarct, and persistent S waves in the lateral precordial leads.

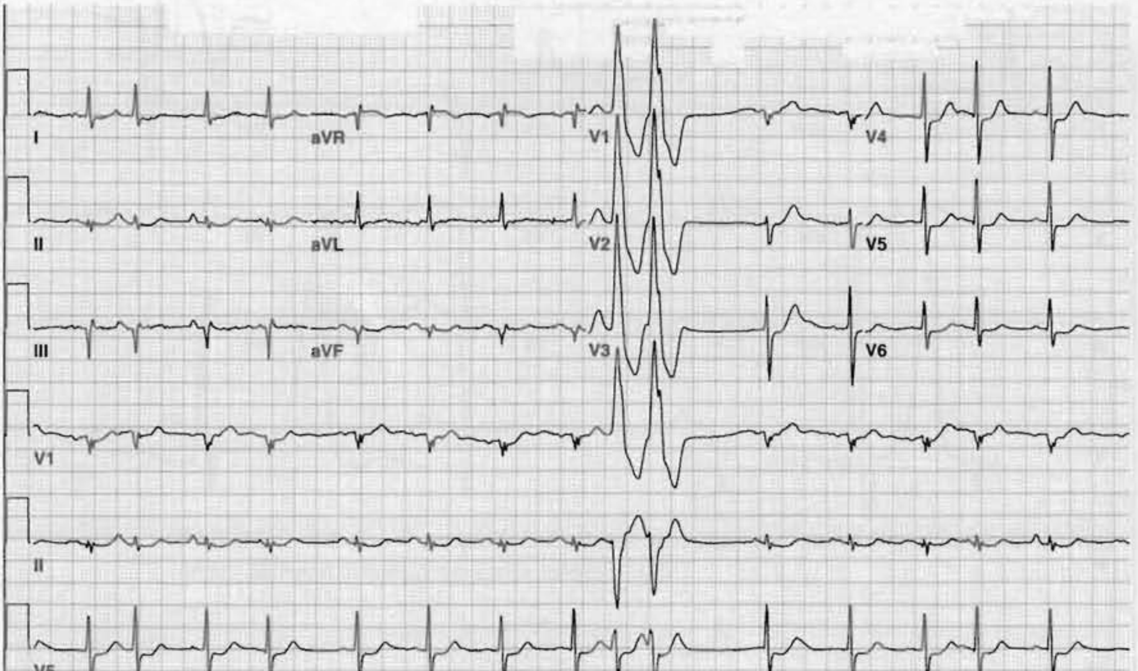


FIGURE 13.3

Rate: The rhythm is very irregular with an average rate of about 90 bpm.

P: The P waves are upright in lead II, more than one P wave morphology is seen, and some QRS complexes do not appear to be preceded by P waves. This would be a good point to determine why these things are so. It might help to first establish what the normal P wave morphology is. Inspection of the lead II rhythm strip is helpful is establishing the rhythm. Starting with the fifth complex a series of QRS complexes that are regularly occurring and associated with P waves with consistent and normal appearance and consistent PR intervals is seen. This is the underlying rhythm. Having established the normal R-R interval and P wave morphology, it becomes evident that several of the QRS complexes in the rhythm strip appear early. Some of them have narrow QRS complexes that look like the normal QRSs in this lead and are preceded by P waves that do not look like the normal P waves; these are PACs. The second QRS complex in this strip has a narrow QRS, but does not appear to be preceded by a P wave. Careful inspection of the T wave after the first QRS complex shows that the "missing" P wave is sitting on top of this T wave, so this is also a PAC. Toward the middle of the rhythm strip two consecutive, early, wide, and bizarre appearing QRS complexes not preceded by P waves appear; these are premature ventricular complexes (PVCs), and since two with similar appearance occur consecutively this is a "uniform couplet." Leads I, III, and aVL show an irregular baseline that appears similar to that seen with atrial fibrillation (A-Fib). Combined with the irregularity of this strip, it might seem reasonable to suspect A-Fib; however, given the regular appearance of P waves and the explanation of the rhythm irregularities due to PACs and PVCs it becomes apparent that this is not the rhythm. The baseline irregularities seen in these leads are from artifact produced by poor skin preparation prior to placement of the electrodes or perhaps from patient tremor. The three leads that show this artifact all share the left arm electrode, so it might help to replace this electrode or establish if a left upper extremity tremor is present.

QRS-T: With the exception of the two PVCs, the QRS complexes are of a normal duration. The axis is approximately −30 and the r waves are taller than the s waves are deep in lead II, so the axis may be considered normal. Significant Q waves are seen in the inferior leads. The lateral precordial leads (V_5 and V_6) show S waves.

Figure 13.4 shows normal sinus rhythm with a second-degree atrioventricular (AV) block with 2:1 conduction, normal QRS configuration, and s waves present in V_5 and V_6.

Rate: The ventricular rate is slow and regular with a rate of about 35 bpm. The atrial rate is about 70 bpm.

P: Two P waves with consistent morphologies are associated with each QRS complex and the PR intervals of the P waves directly preceding each QRS are consistent.

QRS-T: The QRS complexes are narrow and have normal appearance with the exception of persistent s waves in the lateral precordial leads. The axis is approximately 0 degrees.

Comment: With 2:1 second-degree AV block it is not always possible to discern if it is a Mobitz type I or a Mobitz type II and therefore may simply be called a second-

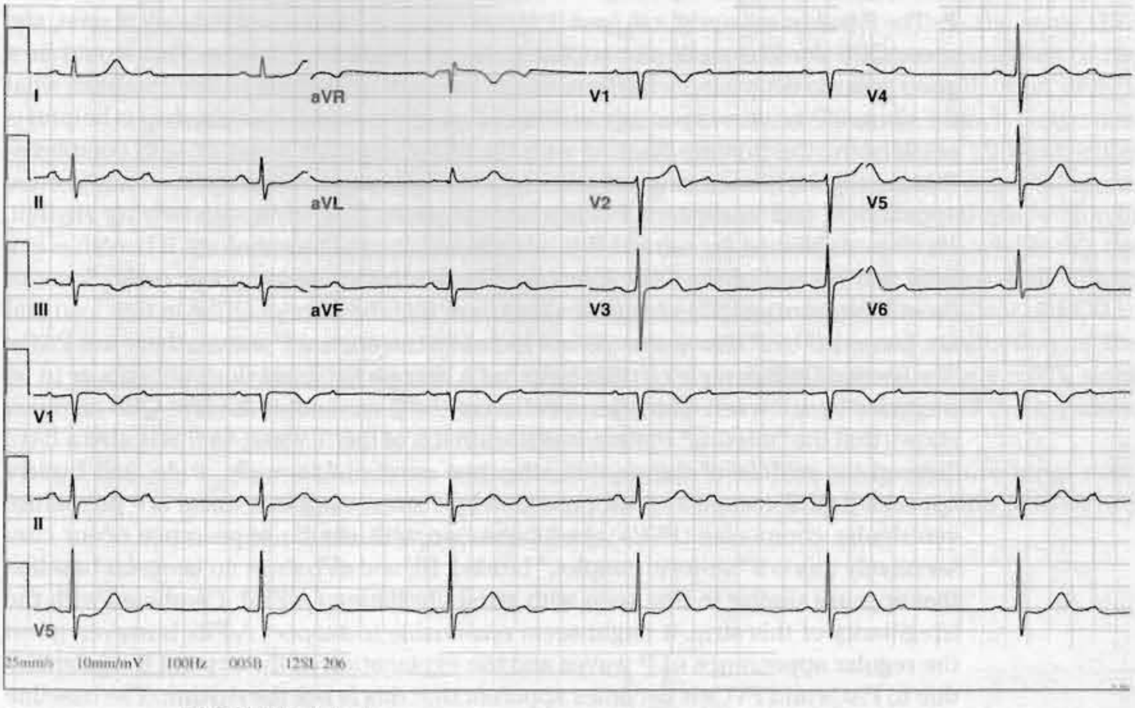


FIGURE 13.4

degree 2:1 block. Longer observation may reveal occasional 3:2 block with lengthening PR intervals establishing a diagnosis of Mobitz type I.

Figure 13.5 shows an atrioventricular nodal re-entrant tachycardia, left anterior fascicular block, poor R wave progression, and possible inferolateral wall ischemia.

Rate: The rate is fast and very regular at about 188 bpm.

P: P waves may not be present, or negative P waves may be appearing after the QRS (e.g., lead II). In either case AV nodal re-entrant tachycardia (AVNRT) would be the presumed rhythm. Atrial abnormalities and PR intervals cannot be determined.

QRS-T: No abnormal Q waves are present, the QRS complexes are narrow with septal q waves in leads I and aVL, and the axis is approximately −60 degrees, indicating left anterior hemiblock. Left axis deviation is implied in the diagnosis of the hemiblock. The r waves do not increase in height in a normal fashion from the right to the left chest leads. The ST segments are significantly depressed in leads II, aVF, V_5, and V_6, and nonsignificant depressions occur in other leads. These depressions may indicate ischemia caused by the increased oxygen demands of the heart associated with the fast rate or may just indicate repolarization abnormalities associated with the fast rate. Some leads (e.g., lead II) appear to have down-sloping ST depression, but this appearance may be due to a negative P wave occurring in the ST segment.

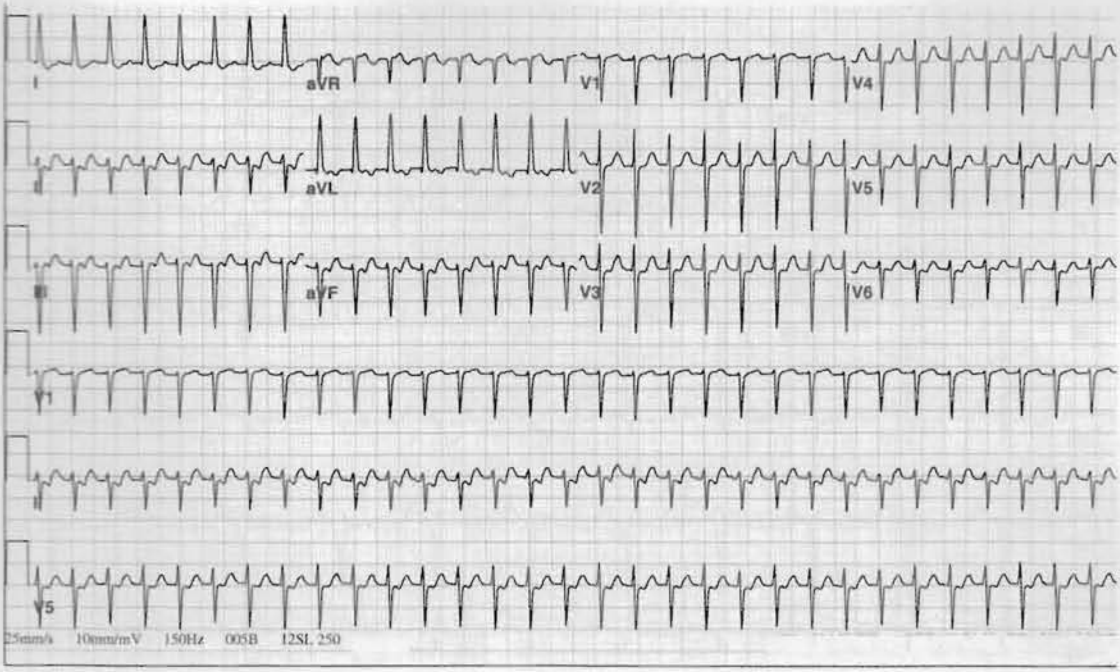


FIGURE 13.5

Figure 13.6 shows a normal sinus rhythm and Wolff-Parkinson-White (WPW).
Rate: Regular at about 70 bpm.
P: The P waves are of modest size, but positive in leads II, III, and aVF. The P waves in lead II may appear to be biphasic, but the negative deflection is actually the beginning of the QRS complex. This can be established using the rhythm strips by comparing where the QRS complex begins in lead V_5 (where it is obvious) with the same time point in the lead II rhythm strip (bottom three lines). Having established where the QRS begins in lead II it becomes apparent that the PR interval is short. A short PR interval should be a signal to look for causes such as WPW.
QRS-T: The QRS duration is prolonged and several leads (e.g., I, V_2) show the distinctive "delta wave" slurring at the beginning of the QRS. This establishes the triad (short PR, wide QRS, delta wave) of WPW. Left axis deviation (−60 degrees) is present, so it might be reasonable to assume a left anterior hemiblock; however, due to the abnormal ventricular activation associated with WPW the usual criteria for hemiblocks cannot be used. Leads III and aVF may appear to have Q waves, but in fact tiny R waves are present. The ST segments are elevated in several leads and the T waves are inverted in leads I and aVL. This is a "pseudoinfarction" associated with WPW, not an actual infarct.

Figure 13.7 shows a normal sinus rhythm, right atrial abnormality, old septal wall infarct, prolonged QT interval, and diffuse nonspecific ST-T abnormalities.
Rate: Regular at about 91 bpm.

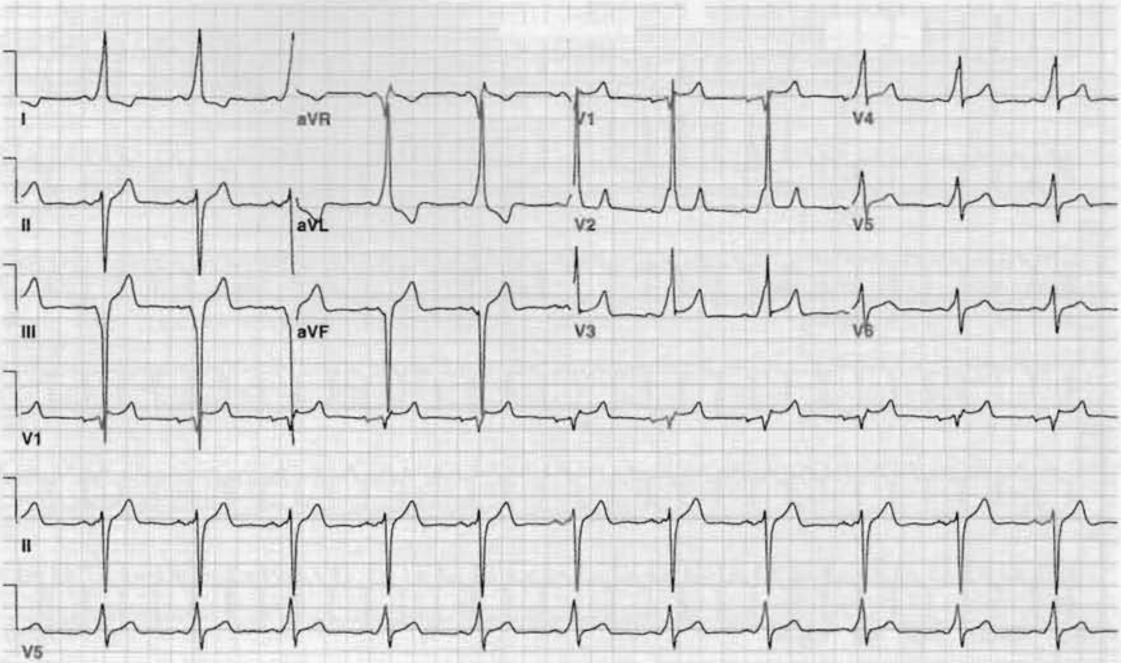

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V5

FIGURE 13.6

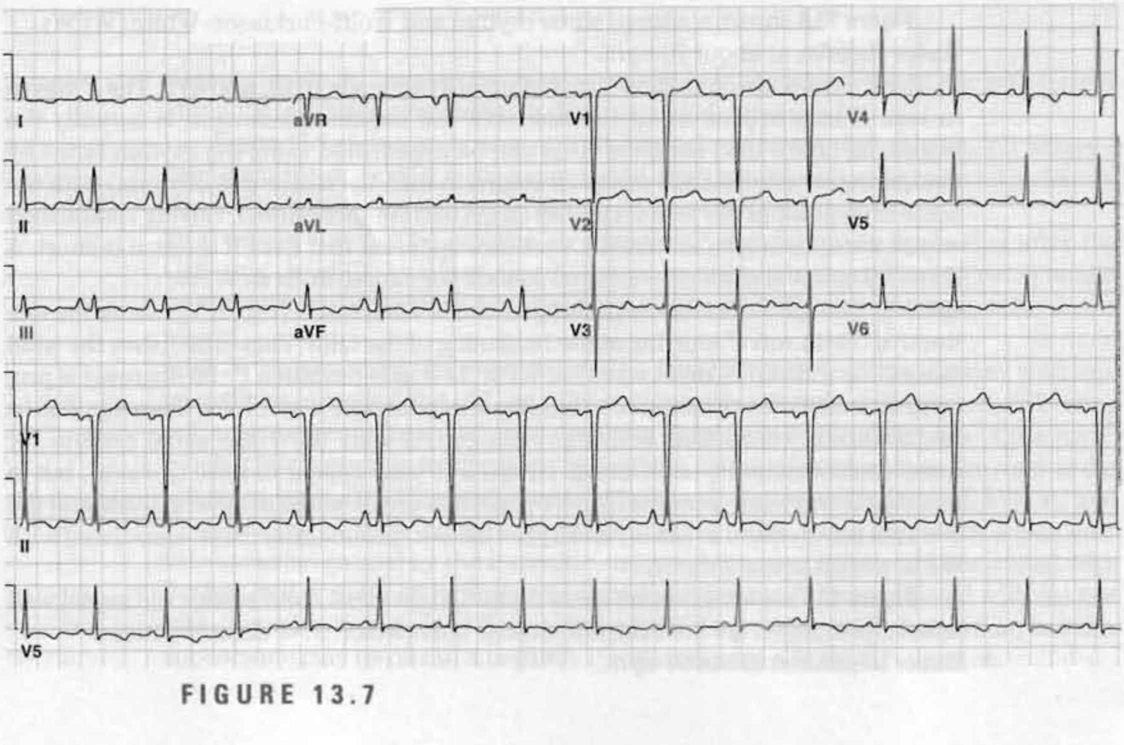

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V5

FIGURE 13.7

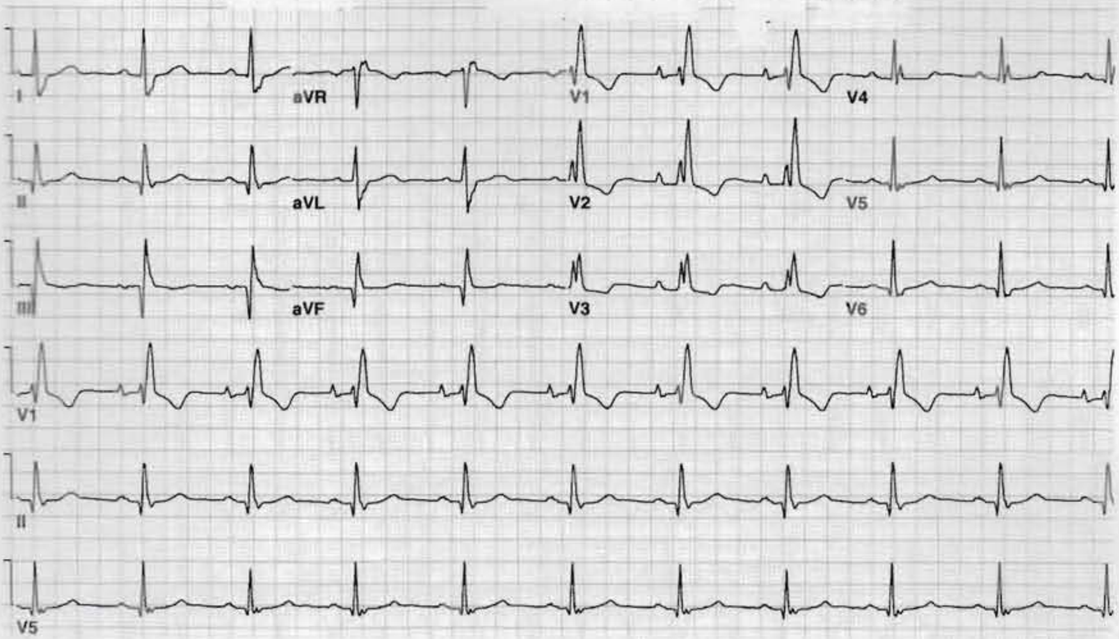


FIGURE 13.8

P: The P waves are taller than 2.5 mm in lead II, indicating right atrial abnormality. **QRS-T:** The axis is normal and about +60 degrees. The QT interval appears to be more than half of the R-R interval, indicating possible prolongation of the QT. The rate-corrected QT interval (QTc) is >440 ms (approximately 475 ms), confirming a prolonged QT interval. Significant Q waves are present in V_1 and V_2, indicating an old infarct of the septum. The modest ST segment elevations in the septal leads (V_1 and V_2) are consistent with the diagnosis of old septal wall infarct. Mild T wave inversions are present in numerous leads, but the shape and depth of these inversions is not specific for any particular abnormality within the context of this ECG.

Figure 13.8 shows a normal sinus rhythm, complete right bundle branch block, and old inferior wall infarct.

Rate: Regular at about 62 bpm.

P: The P wave morphology in lead II is normal, and one P wave is associated with each QRS in a normal fashion.

QRS-T: The axis is normal at about +60 degrees. The QRS duration is >120 ms in some of the limb leads. This should instigate a search for left bundle branch block (LBBB) or right bundle branch block (RBBB). An rSR' is present in V_1 and V_6 shows an S wave; therefore, an RBBB pattern is present. Significant Q waves

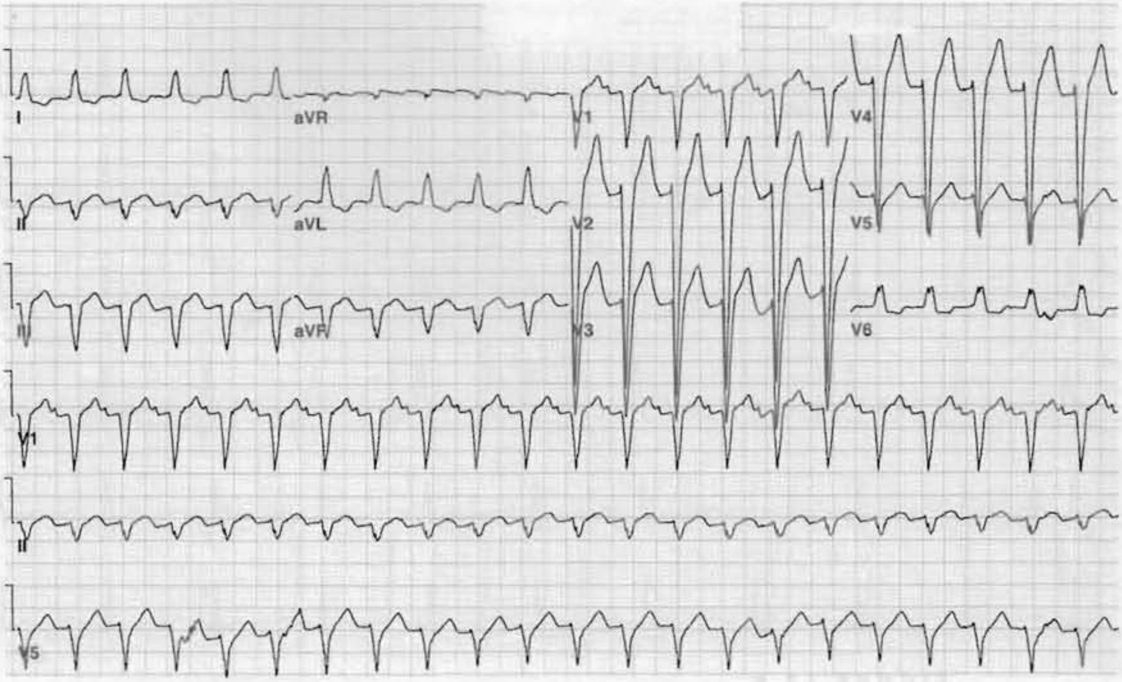


FIGURE 13.9

appear in all of the inferior leads, suggesting an old infarct. ST segment depression and T wave inversions are present in the right chest leads secondary to the RBBB.

Figure 13.9 shows sinus tachycardia versus sinoatrial (SA) nodal re-entrant tachycardia versus ectopic atrial tachycardia, complete left bundle branch block.

Rate: Very regular at about 136 bpm.

P: The P wave morphology in lead II and the other inferior leads is difficult to discern as the P waves appear to be merging with the preceding T waves; P waves are clearly visible in V_1 and have a normal relationship to the QRS complexes. The very regular rhythm and the rate would favor a diagnosis of SA nodal re-entrant tachycardias instead of sinus tachycardia (although it could be a sinus tachycardia). If the P waves are not positive in the inferior leads (difficult to tell in this ECG), ectopic atrial tachycardia is favored.

QRS-T: The axis shows left axis deviation (LAD) at about −60 degrees. The QRS duration is >120 ms in some of the limb leads. This should instigate a search for LBBB or RBBB. A wide R is present in V_6 and a QS pattern is seen in V_1; therefore, an LBBB pattern is present. ST segment elevations are present in the right chest leads secondary to the LBBB.

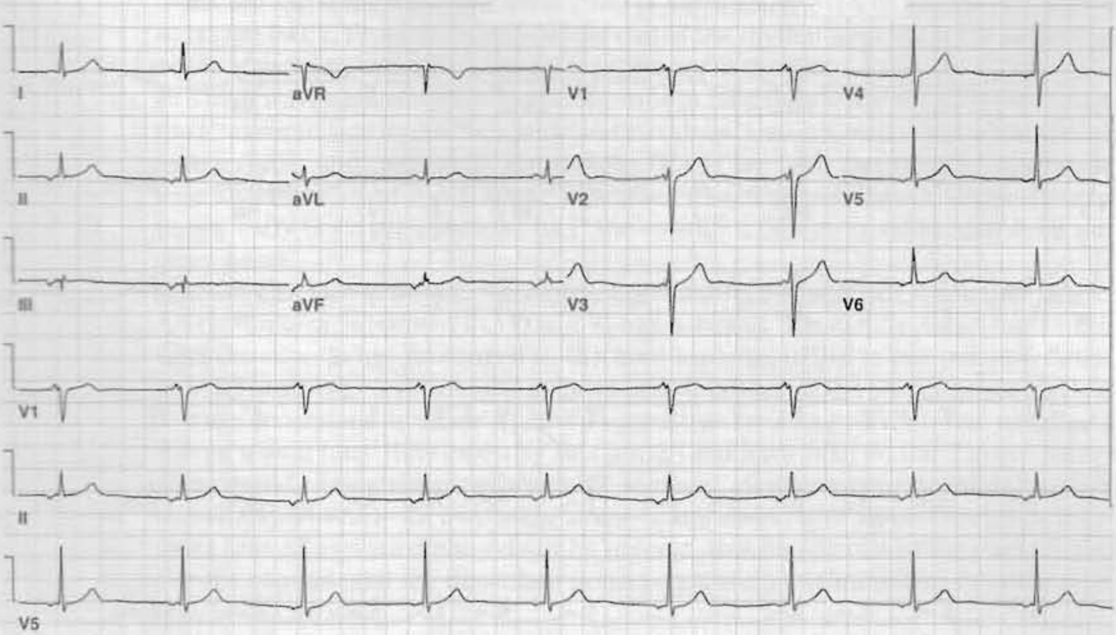


FIGURE 13.10

Figure 13.10 shows a junctional escape rhythm (ectopic atrial bradycardia) and normal QRS complex configuration.

Rate: Regular at about 52 bpm.

P: The P waves are negative in the inferior leads, implying a junctional rhythm.

QRS-T: The axis is normal at about +30 degrees. The QRS complexes are normal in amplitude, duration, and appearance. The ST segments and T waves are normal.

Figure 13.11 shows atrial fibrillation with moderate ventricular response and acute inferior and anterolateral wall infarct.

Rate: Very irregular at about 90 bpm.

P: Regularly occurring, organized atrial activity is not present, and the ventricular rhythm is grossly irregular.

QRS-T: The axis is normal at about +30 degrees. Significant Q waves are present in the inferior leads. The ST segments are significantly elevated in the inferior leads and V_3 to V_6, indicating an acute STEMI; the ST segment is significantly depressed in aVL likely due to remote ischemia.

Figure 13.12 shows a normal sinus rhythm with first-degree AV block and frequent premature atrial complexes sometimes occurring in a bigeminal pattern, biatrial abnormality, left anterior fascicular block, and evolving ST segment elevation infarct or ischemia.

Rate: The rate is normal (77 bpm); some beats occur early. What is causing the early beats can be explored here. Note that the P wave morphologies of the early

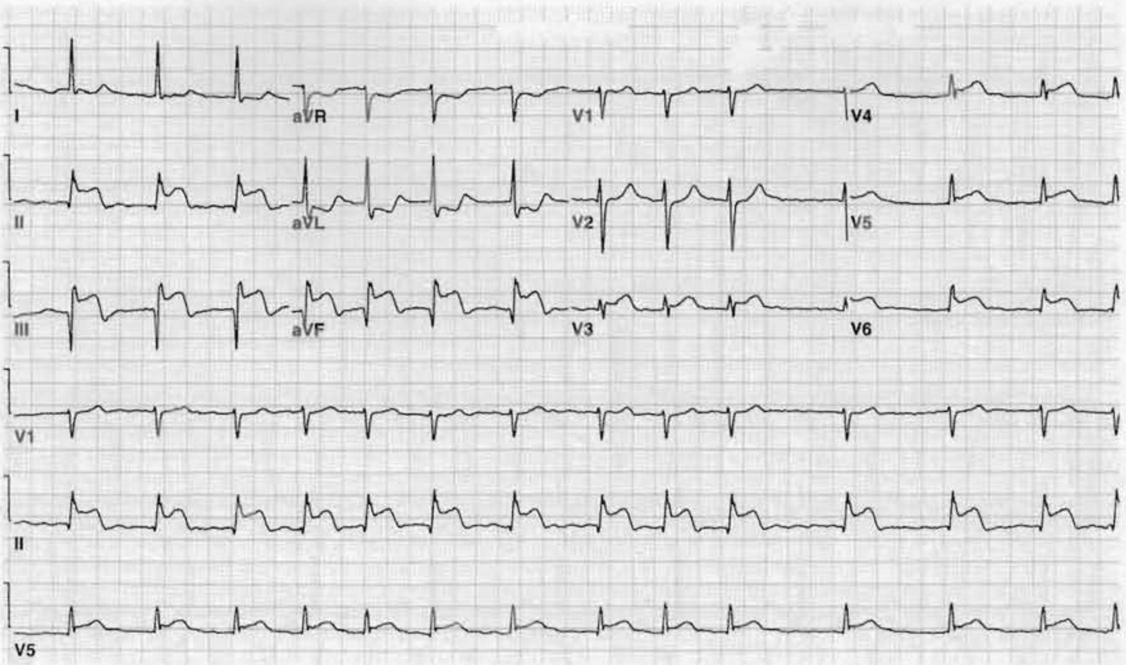

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V5

FIGURE 13.11

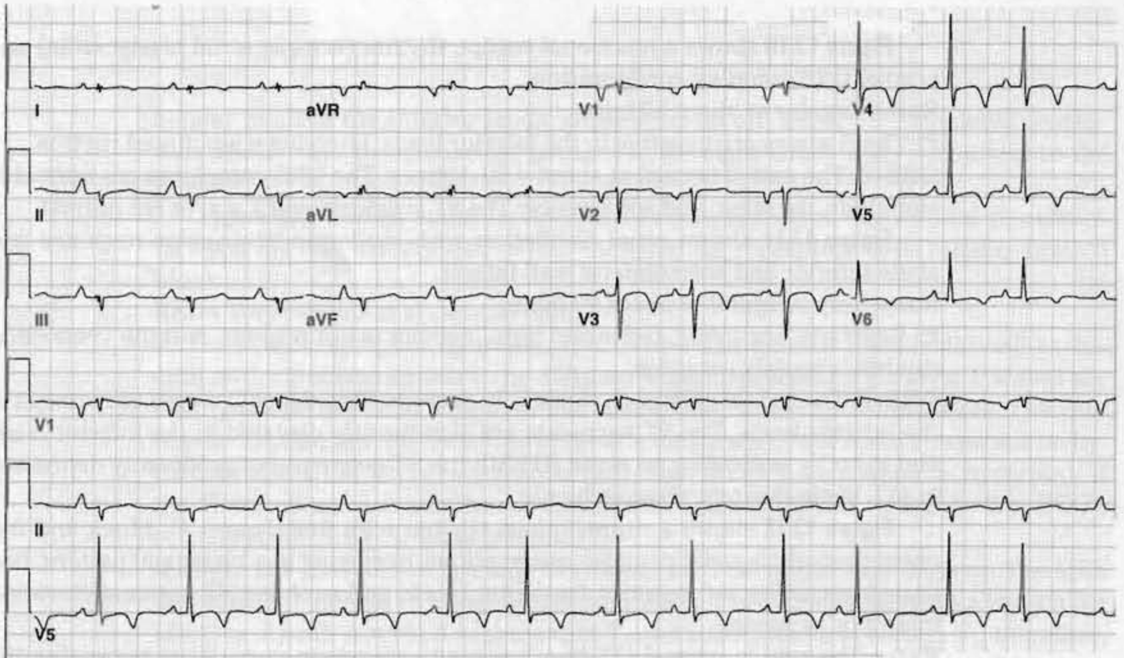

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V5
25mm/s 10mm/mV 40Hz 005B 12SL 206 CID: 3

FIGURE 13.12

beats are different from the norm (this is particularly evident in lead V_1), these beats are PACs. From the middle to the end of the rhythm strip PACs are occurring every other beat, thus atrial bigeminy is present at that time.

P: The P waves are abnormally tall and wide in lead II (and the negative portion of the P waves in lead V_1 is a "box wide and box deep"), so criteria for left atrial abnormality (LAA) and right atrial abnormality (RAA) are both met (biatrial abnormality), The PR interval is >200 ms, indicating first-degree AV block. As previously noted, early beats are preceded by P waves with different morphologies from the other beats.

QRS-T: The axis is about −90 degrees, indicating left anterior fascicular block. An rsr' pattern is present in V_1, and V_6 has s waves; this can be noted but as the QRS duration is not prolonged in the limb leads an RBBB is not present. Some mild ST segment elevations are present and the shape of the ST segments and the T wave inversions in leads V_2 to V_6 suggest an evolving STEMI; but usually Q waves would have developed by this point (and have not) so the T wave inversions could indicate ischemia/non-ST segment elevation myocardial infarction (NSTEMI). This may be confusing as the designation NSTEMI pretty clearly indicates that ST elevation should not be present; this case is one of the exceptions. For the physiologist the important point is that ischemia/infarction of some type may be occurring.

Quiz 13

Describe the rhythm shown in the following strips.

1. ______________________________

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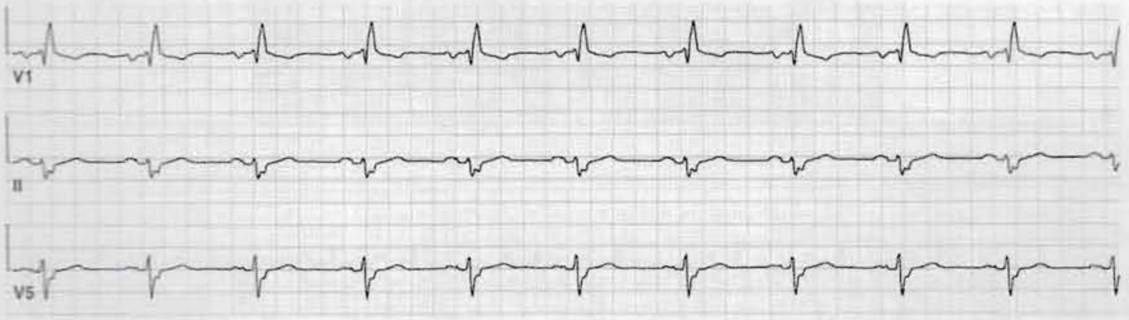
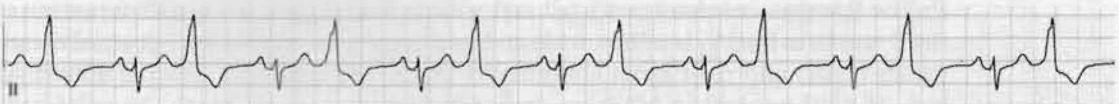
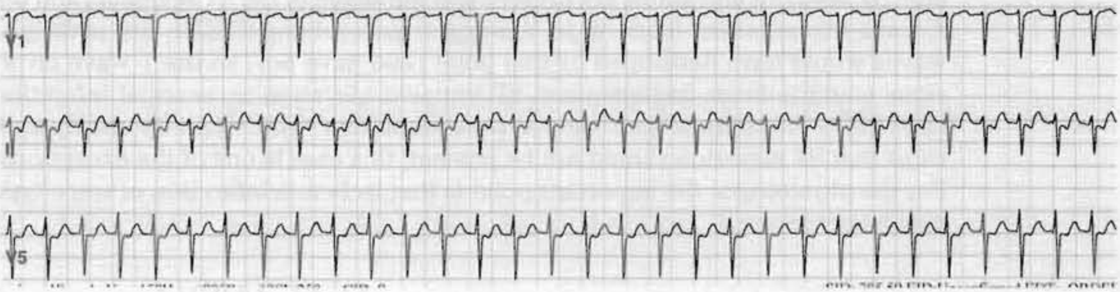


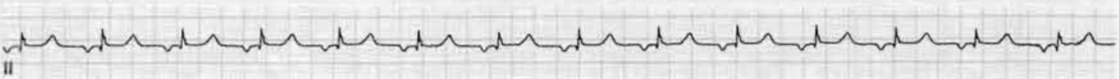
2.



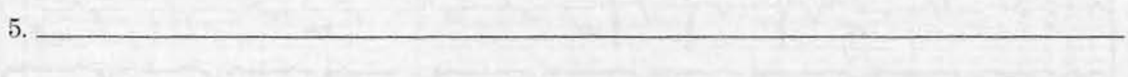
3.



4.



5.



6. ______________________________

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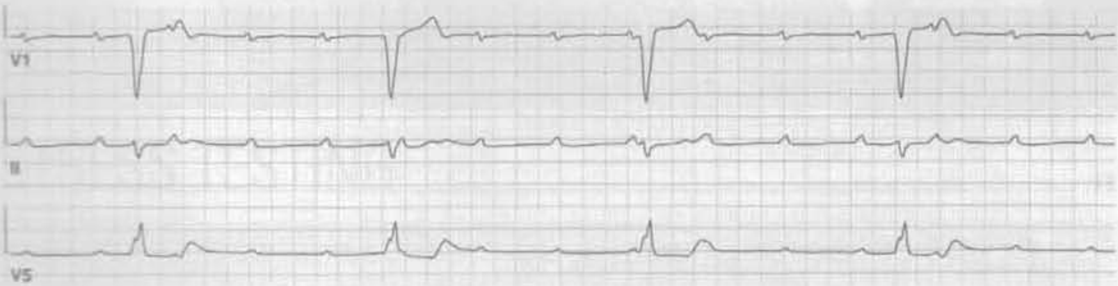

7. ______________________________

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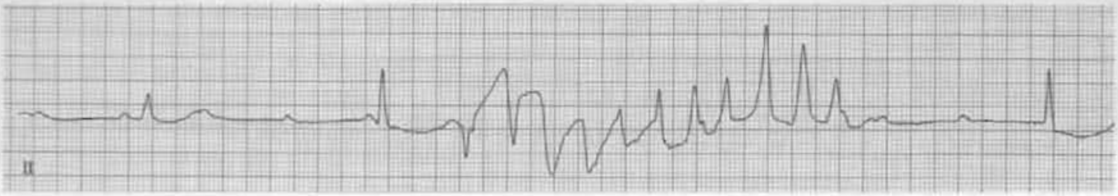

8. ______________________________

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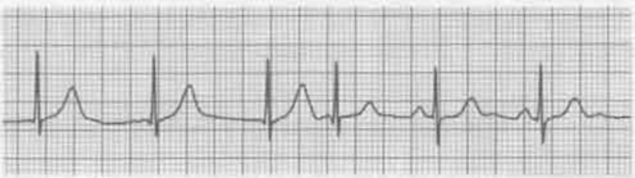

9. ______________________________

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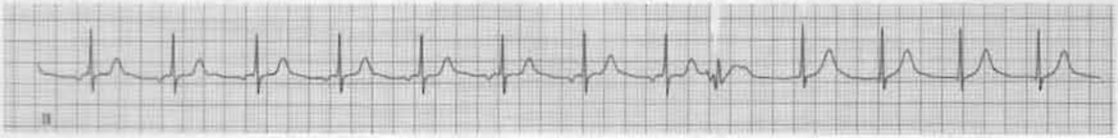
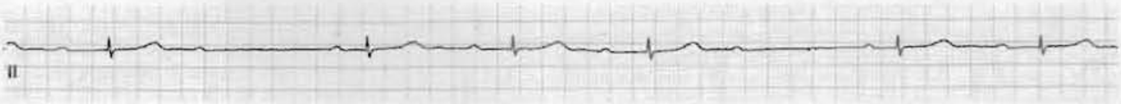
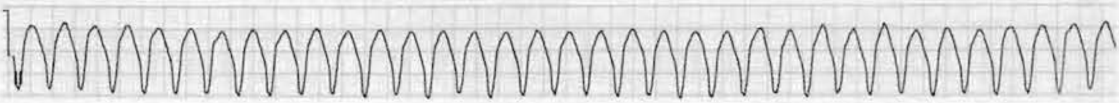


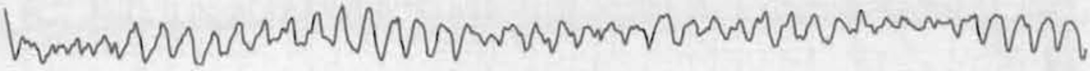
10. ______________________________

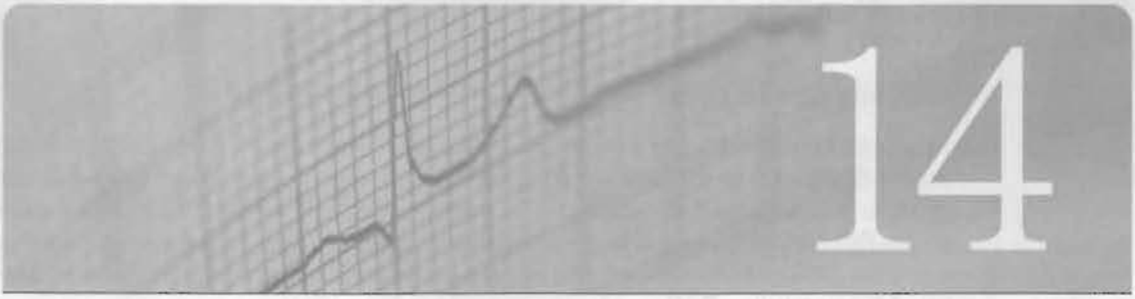


11. ______________________________



12. ______________________________





14

STRESS TESTING

- Prestress ECGs
- Technical Problems
- Inspection of the Resting ECG
- Exercise ECGs
- ST Segment Changes
- ST Depressions Only Occurring in the Inferior Leads
- T Wave Inversions
- Arrhythmias
- ST Segment Elevations
- Non-ECG Stress Testing Variables

In many aspects, the interpretation of ECGs in the setting of a *stress test* is no different from reading an ECG in any other arena. However, some matters do warrant special attention. In this chapter, we review some considerations pertinent to various types of stress tests.

Prestress ECGs

It has been noted in earlier chapters that unless otherwise indicated, an ECG is assumed to have been recorded with the patient supine and at rest with the electrodes in the normal positions. Prior to exercise it is best to run properly labeled resting 12-lead ECGs under the three conditions described below.

Supine (with standard electrode positioning)

As it is taken under standard conditions, the supine resting cardiogram can be fairly compared with previous and future resting ECGs. For pharmacological stress tests (e.g., dipyridamole) this is the only resting ECG needed as the patient will remain in the supine, resting position throughout the test.

Supine Mason-Likar

For exercise tests the limb electrodes must be moved onto the torso as described in Chapter 7. In some patients the Mason-Likar ("exercise") electrode placement causes changes on the ECG; comparison of this cardiogram with the standard supine ECG will establish which if any changes are caused by electrode positioning. In many patients no changes will occur.

Standing Mason-Likar

In some patients standing causes ECG changes; comparison of this ECG with the supine Mason-Likar will test for such changes. In many patients the ECG will not change. For treadmill exercise this is an appropriate baseline ECG to compare stress ECGs to when evaluating ST segment changes. For cycle ergometer exercise a resting ECG performed with the patient seated on the ergometer may be substituted for a standing ECG.

Technical Problems

If artifact (Chapter 12) is present at rest it almost certainly will worsen with exercise. Many modern ECG machines have a built-in system for testing if the "prep" was properly done. This may be of help, but tracings should also be inspected manually for technical adequacy. If needed, reprepping or other measures to correct technical problems should be done prior to exercise. Once the technical problems are resolved the resting strip(s) should be rerun.

Negative P waves in lead II or other unusual findings such as severe axis deviation should initiate a check of wire placement, as it is common to misplace wires (e.g., the LA wire placed on the RA electrode). It is also common for resting tracings to have artifact from patient movement, coughing, and so forth. If this occurs the source of the artifact should be identified and corrected and a "clean" tracing obtained prior to proceeding.

Inspection of the Resting ECG

It is important to inspect the resting ECGs prior to initiating stress. As per the American College of Sports Medicine (ACSM) and other organizations, certain resting ECG findings (e.g., significant change suggesting ischemia) are contraindications to stress testing. Contraindications are covered in *ACSM's Guidelines for Exercise Testing and Prescription*; it is recommended that those who perform stress testing be thoroughly familiar with this resource.

Other resting findings, while not contraindications to testing, are important to note. For example, left bundle branch block, left ventricular hypertrophy with "strain," Wolff-Parkinson-White, and digitalis effect can cause resting ST segment depressions and/or T wave inversions that render the exercise ECG nondiagnostic. The resting changes resemble those seen with ischemia, so it is difficult or impossible to tell if ischemia occurs during the stress test. Even if additional ST segment and/or T wave changes occur with stress, the usual diagnostic criteria are not considered valid. Ischemia may still be evaluated if an imaging modality is used as in nuclear stress testing or exercise echocardiography, so if a "plain" stress test (exercise with ECG only and no imaging) was ordered, it may be wise to consult with the medical director before proceeding with the test. Useful information such as functional capacity, heart rate, and blood pressure responses can still be obtained from a plain stress test, but in many instances a different type of test (e.g., nuclear stress) will be appropriate, particularly if the purpose of the test is to diagnose coronary disease.

Exercise ECGs

Most modern systems will automatically label ECGs, but in some cases it must be done manually. When a cardiogram is recorded during exercise the type and intensity of exercise and the time should be recorded. For example "Treadmill 1.7 mph/10% grade 2:00 minutes" or "Cycle ergometer 50 watts stage time 2:00 minutes, total exercise time 4:00" make it clear what the conditions were when the ECG was recorded. For pharmacological tests ECGs should be labeled with the protocol time and dosage and type of medication. For example, "Dobutamine, minute 6:00, 20 µg/kg/min." Cardiograms recorded during the postexercise period should also be labeled with specific information. For example, "2:00 recovery" is a common way to indicate that the recording was made 2 minutes into the postexercise (or other stress) period.

Although minimized by good skin preparation, even under the best of circumstances some motion artifact will often be present on the ECG during exercise stress tests. It is important that at least one lead give a "clean" enough tracing to monitor the rhythm. Typically three leads will be monitored continuously with 12-lead strips run at predetermined intervals and additional rhythm strips printed as needed. Commonly leads II, V_1, and V_5 are monitored continuously as this allows "views" of the inferior, septal, and lateral walls, respectively. Additionally, lead II is in line with the usual P wave axis, assisting with rhythm monitoring, and V_1 and V_5 are well positioned to detect rate related bundle branch blocks. Monitoring these three leads permits a high likelihood that important changes during stress will be observed in at least one of them. Other leads may be used. For example, if it appears that the ST segments are becoming depressed in V_5 it may be useful to monitor, at least temporarily, other lateral leads.

The ECG machine will usually be programmed to print 12-lead ECGs every minute (or minimally every stage) and at peak exercise during stress and every 2 to 3 minutes during recovery. If ectopic beats or other events of interest occur, additional strips (often three-lead rhythm strips) should be run as needed. The 12-lead strip run at peak exercise often shows a great deal of motion artifact, so it may be useful to run an additional 12-lead or long three-lead rhythm strip shortly after peak. As the patient enters the recovery period motion artifact usually is reduced, but any ischemic changes should still be present.

ST Segment Changes

The interpretation of ST segment changes is of great interest during stress testing. An artery significantly narrowed by coronary artery disease (CAD) is often still capable of supplying enough blood and, therefore, oxygen to meet the metabolic demands of the heart at rest, so the resting ECG in such patients may be perfectly normal. Adding stress via exercise or other mechanisms increases the metabolic demands on the heart. A narrowed coronary artery may be unable to supply sufficient blood in this setting, rendering the subendocardium ischemic with resultant ST segment depressions.

In order to quantify ST segment changes a baseline must be established. As described in Chapters 1 and 11, the PR or TP segments can be used as baselines for ST segment measurements. During stress testing it is recommended that the PR segment be used. The typical ECG finding indicating myocardial ischemia during stress is significant depression of the ST segment. The most common definition of "significant" depression is an ST segment that is 0.1 mV (1 mm) or more below baseline. Depressions of less than 0.1 mV (1 mm) may be noted, but are not usually considered indications of a "positive" (indicating the presence of disease) test. As the patient is moving during an exercise stress test motion artifact often complicates interpretation. For example, sporadic artifactual ST depressions often appear. Because of this it is common to require three or more consecutive complexes showing ST depression to accept it as a true finding.

ST segment depressions occur in three patterns shown in **Figure 14.1**. If the ST segment is depressed but flat it is called *horizontal depression* (Fig. 14.1A), depression that becomes greater over the course of the ST segment is called *down-sloping* (Fig. 14.1B), while depression that lessens over the course of the ST segment is termed *up-sloping* (Fig. 14.1C). With horizontal depression it does not matter where the depth of the depression is measured as it will yield the same result, but with up- or down-sloping depressions the magnitude of ST depression will vary depending on the measurement point. One commonly used measurement point is 80 ms (0.08 sec) after the J point (where the QRS complex ends and the ST segment begins). Other measurement points are in use, so it is good policy to not only state the magnitude of the depression but also where the ST measurement was made. For example one might describe a particular ST segment as having "0.2 mV of down-sloping ST depression 80 ms post J point." It is common to use mm instead of

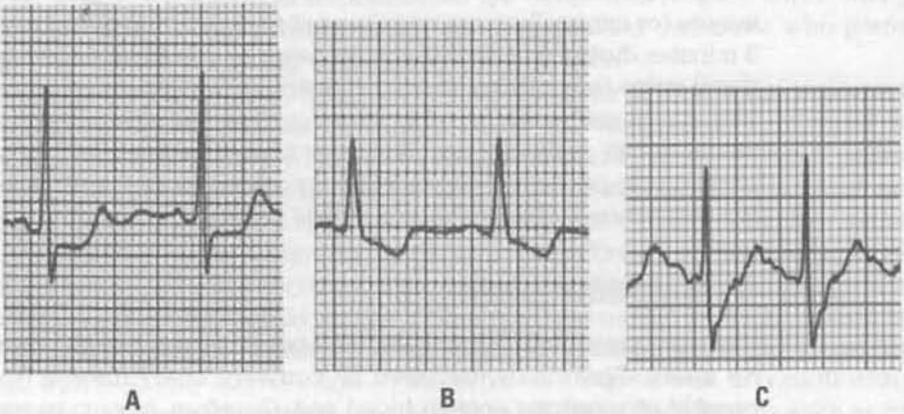


FIGURE 14.1 Patterns of ST segment depression: horizontal (**A**), down-sloping (**B**), and up-sloping (**C**).

mV, sec instead of ms, and "post QRS complex" instead of "post J point," so the same findings could be described as "2 mm of down-sloping ST segment depression 0.08 sec post QRS complex."

Using the terminology just described, the ST changes shown in Figure 14.1 could be reported as follows:

a. "0.1 mV (or 1 mm) of horizontal ST segment depression"
With horizontal depression it is not critical to state where it was measured as the value is the same at all points of the ST segment.

b. "0.15 mV (or 1.5 mm) of down-sloping ST segment depression 80 ms post J point"
The amount of depression varies depending on where it is measured. At a point 80 ms (two small boxes) past the end of the QRS complex the ST segment is about 1.5 mm below the PR segment baseline.

c. "up-sloping, but isoelectric 80 ms post J point"
Initially the ST segment is depressed, but with up-sloping changes the depression lessens over time, and in this instance it has essentially returned to the baseline by 80 ms post-QRS complex. ST segments on the baseline are often referred to as isoelectric.

Figure 14.2 shows resting and stress ECGs from a treadmill exercise test. At rest the ST segments in all leads were isoelectric. During exercise ST depression occurred in multiple leads. A complete description of the results of this test should include a description of the magnitude of the changes, which leads they occurred in, and what exercise intensity elicited the changes. Often ST depression occurs during exercise. During this test the patient exercised to an estimated intensity of 6.2 METs (metabolic equivalents), but the ST segments did not become depressed until the recovery (postexercise) period. Thus, these ECG changes could be described as "0.1 mV of down-sloping ST segment depression in the inferior leads and V_4 to V_6 during recovery from 6.2 METs." If changes occur during exercise it is best to describe when they initially became significant and for any worsening. For example, changes that were significant in V_5 and V_6 at 5 METs but worsened as the test progressed might be described as "0.1 mV of horizontal ST segment depression in the lateral precordial leads at 5 METs progressing to 1.5 mV of horizontal depression at 7 METs." It is also important to indicate how long it took for depressed ST segments to return to baseline (e.g., "ST segments returned to baseline 5 minutes into recovery").

ST Depressions Only Occurring in the Inferior Leads

To be considered indicative of a "positive" test, ST segment changes must occur in two or more contiguous leads. In this setting contiguous means leads grouped together and associated with a certain area of the heart. For example, leads V_5, V_6, I, and aVL are contiguous as they are all associated with the lateral wall. Leads II, III, and aVF are contiguous and associated with the inferior wall; ST segment depressions confined to the inferior leads commonly occur in patients who do not have significant coronary artery disease, resulting in a "false positive" (positive test, but the patient does not actually have the disease).

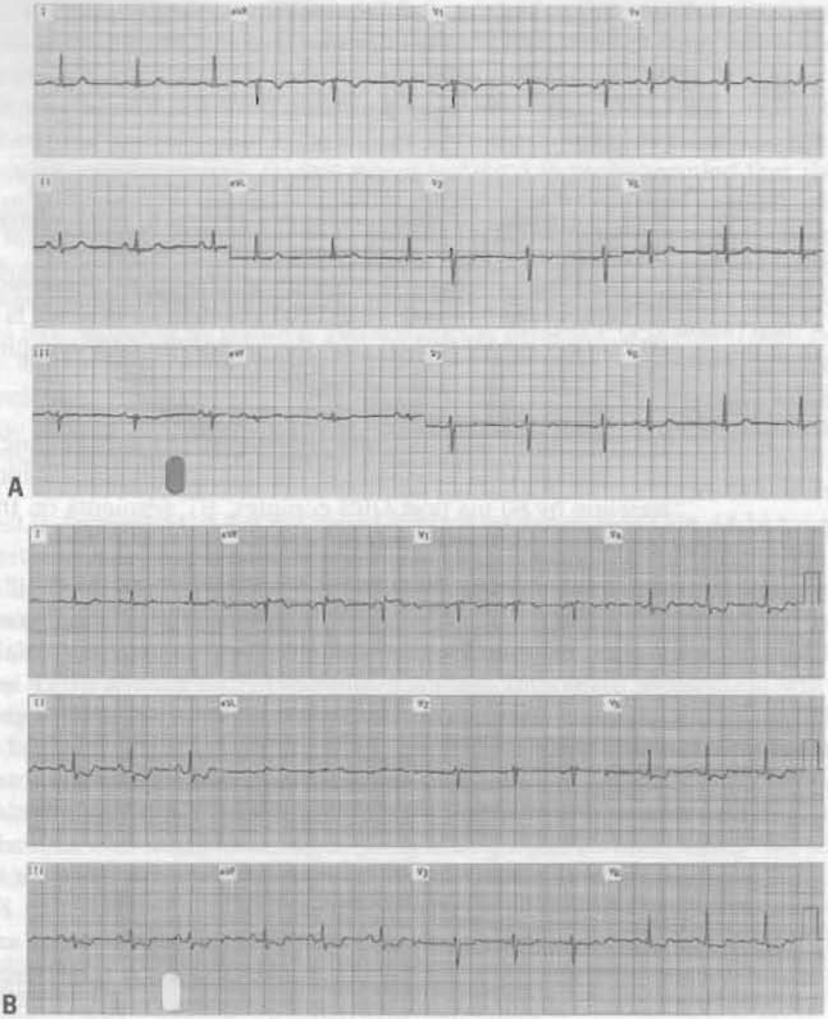


FIGURE 14.2 ST depression during exercise stress. **A:** At rest. **B:** During stress.

T Wave Inversions

Stress induced T wave inversions (**Fig. 14.3**), whether occurring in isolation or accompanied by ST segment depressions, may also be evidence of ischemia. If T wave inversions are not associated with ST segment depressions they are not usually considered strong enough evidence to consider a test "positive," but they should be noted. Because these changes are sometimes caused by hyperventilation, some authorities recommend that after the T waves return to baseline in the recovery period the patient be asked to hyperventilate for a few minutes to see if the T wave inversions reoccur. It they do, presumably ischemia is not the cause. Hyperventilation should be performed with the patient seated as a precaution in case dizziness occurs.

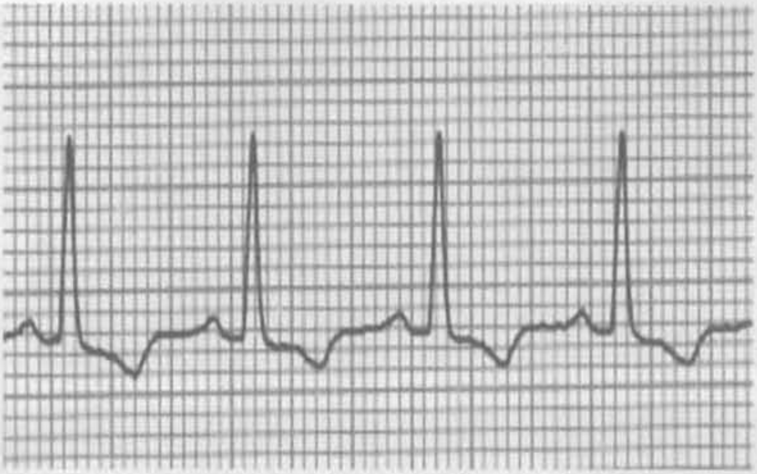


FIGURE 14.3 Stress T wave inversion.

Arrhythmias

The type and relative frequency of arrhythmias should be described. For example, a phrase such as "frequent PVCs including multiform couplets" could be used to describe **Figure 14.4.** The ECG shown in **Figure 14.5** is from a test that was stopped because of the onset of ventricular tachycardia. The report of this test might

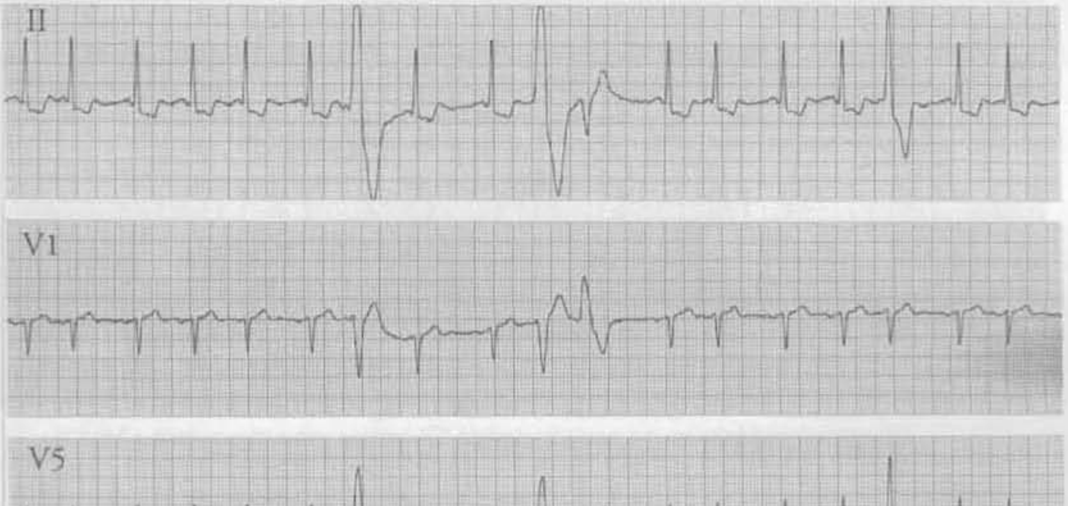


FIGURE 14.4 Arrhythmias during stress.

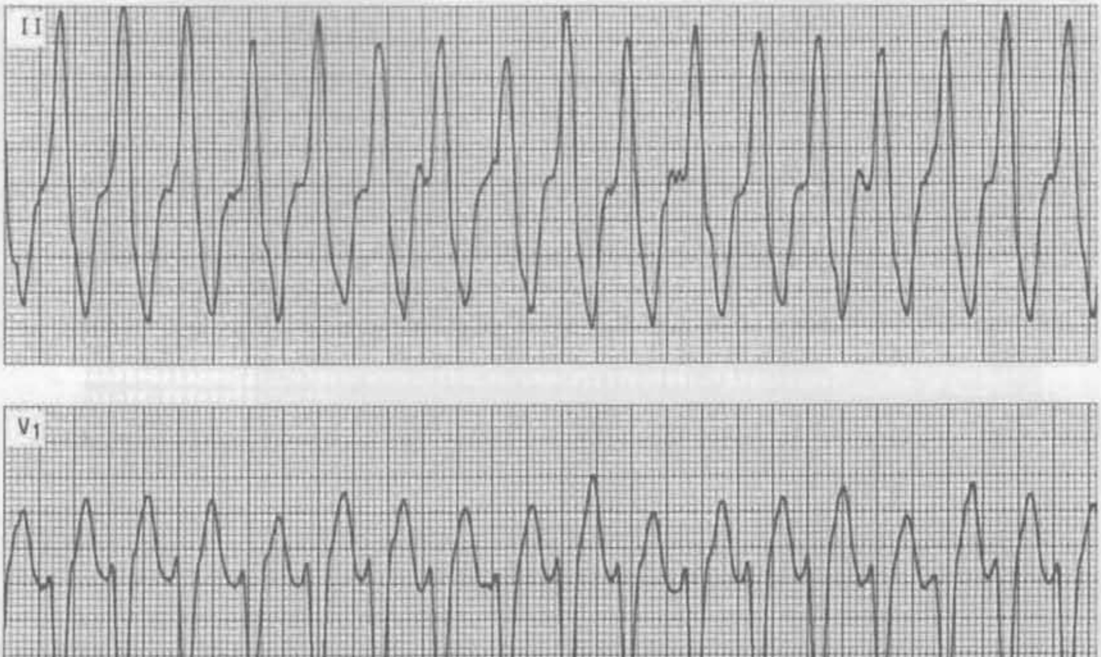


FIGURE 14.5 Ventricular tachycardia during stress.

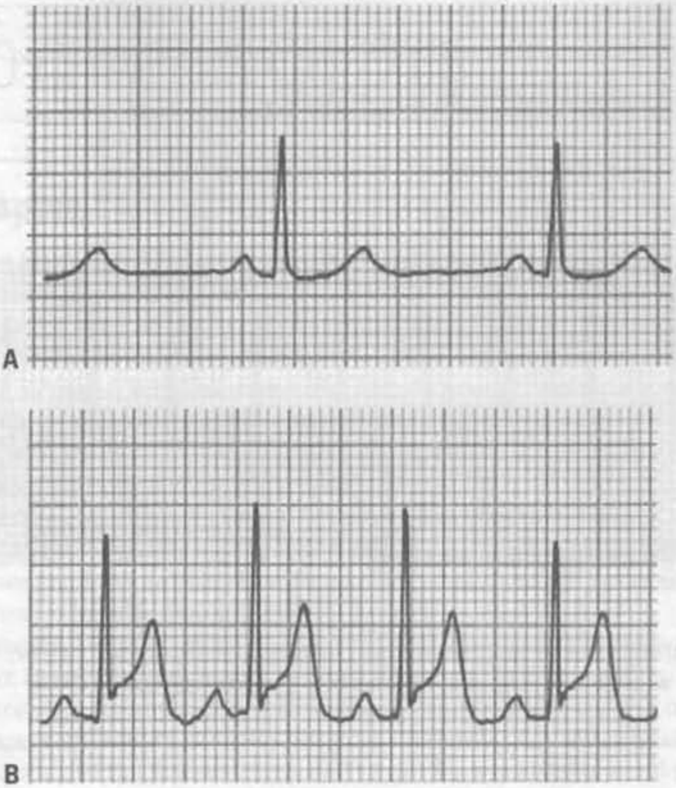


FIGURE 14.6 Stress ST elevation. **A:** At rest. **B:** During exercise.

include a phrase like "the test was terminated due to monomorphic ventricular tachycardia."

ST Segment Elevations

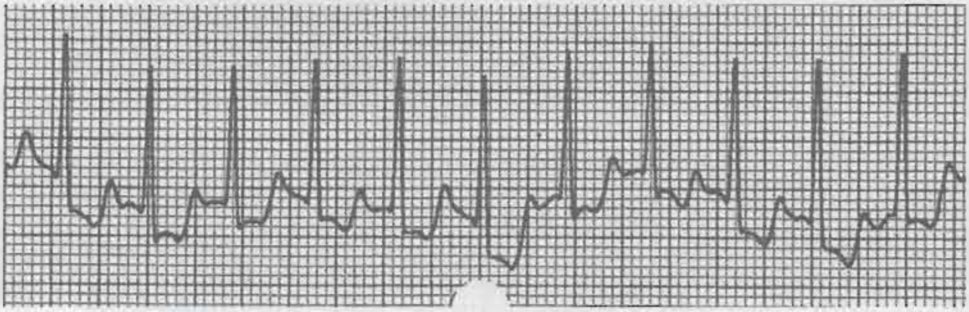
Although rare, elevation of the ST segment (presumably due to transmural ischemia) is sometimes seen during stress and is an indication to stop the test. Coronary angiography later revealed that the patient whose ECG is shown in **Figure 14.6** had a 99% occlusion of a major coronary artery. In addition to severe occlusions of a major coronary artery, ST elevations can be caused by vasospasm.

Non-ECG Stress Testing Variables

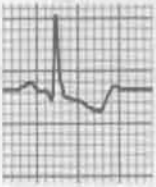
Other factors such as blood pressure responses, exercise capacity, and signs and symptoms offer diagnostic and prognostic information and should also be evaluated during a stress test. The interpretation of these parameters is addressed in *ACSM's Guidelines for Exercise Testing and Prescription.*

Quiz 14

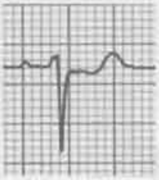
1. Is the ST depression real or motion artifact?



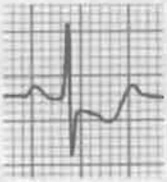
2. Describe the stress ST segments and T waves in A through F below. Assume that the resting ST segments were isoelectric and the resting T waves upright.



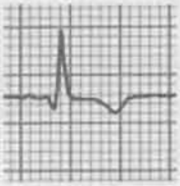
A



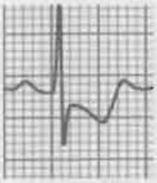
B



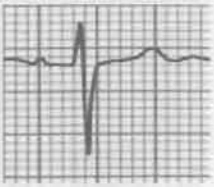
C



D



E



F

Appendix

ANSWERS TO CHAPTER QUIZZES

Chapter 1

1. (a) The QRS consists solely of a fairly large positive deflection, thus it could be described as a large R.
   (b) A fairly large positive deflection is followed by a fairly large negative deflection or "large R, large S."
   (c) A large initial negative deflection is followed by a relatively small positive deflection resulting in a "large Q, small r."
   (d) A large initial negative deflection is followed by a return to baseline thus "large Q, large S."
   (e) A small initial positive deflection is followed by a deep negative deflection (which since it is not the initial deflection is an S wave rather than a Q wave) and the QRS ends with another small positive deflection yielding a "small r, large S, small r prime."
2. The distance from the very beginning of the P wave to the beginning of the QRS complex is seven small boxes so the PR interval is 280 ms (7 × 40). The distance from the beginning of the R wave to the end of the s wave is one and a half small boxes, therefore, the QRS duration is 60 ms (1.5 × 40). The distance from the beginning of the R wave to the end of the T wave is eight small boxes or 0.32 sec (8 × 0.04, the Bazett formula requires the measurements to be entered in sec, not ms), the distance from the peak of the first R wave to the peak of the second R wave is 21.5 mm, yielding an R-R interval of 0.86 sec (21.5 × 0.04). The square root of 0.86 (rounded to the nearest hundredth) is 0.93, 0.32 divided by 0.93 equals approximately 0.34. A QTc of 0.34 sec (or 340 ms) would be considered normal as it is <0.44 sec (or 440 ms).
3. The distance from the peak of the first R wave to the peak of the second R wave is 21 mm. Dividing 1,500 by 21 yields a HR of 71.43 bpm. This would be the HR during this R-R interval. This would also be a good approximation of the average HR if ensuing R-R intervals (not shown) were consistent.
4. Using the triplets method, the HR is found to be approximately 90 bpm. The R-R intervals are fairly consistent. The first R wave falls on a thick line. The next R wave falls closest to two thin lines past the third thick line. Had it fallen on the third thick line the rate would have been 100 bpm. Had it fallen on the fourth thick line the rate would have been 75 bpm. The rate is therefore between 75 and 100 bpm. The span between 75 and 100 bpm is 25 bpm, and this is represented by the five small boxes, thus each small box represents approximately 5 bpm (25 ÷ 5 = 5). Since the second R wave falls nearest to the second thin line past the thick line representing 100 bpm, the rate must be approximately 10 bpm <100, or 90 bpm.

5. The R-R intervals are not consistent; therefore, a method that averages the rate must be used. Since three second marker lines are visible (bottom of strip), an easy method to use is counting the R-R Intervals (not R waves) for a six second period and multiplying by 10. There are eight R-R Intervals contained within the six second interval, thus the rate is approximately 80 bpm (8 × 10 = 80).

Chapter 2

1. SA node, atrial tissue, AV node, bundle of His, left and right bundle branches, Purkinje fibers, ventricular tissue.
2. Normal sinus rhythm with a junctional premature complex (JPC). Depolarizations originating low in the atria or from the AV junction often result in negative P waves in lead II as the depolarization spreads from bottom to top in the atria and therefore largely toward the negative pole of lead II. Depending on the exact timing, location, and other factors, these beats may result in a negative P wave before the QRS complex (when atrial depolarization occurs before ventricular depolarization), a negative P wave after the QRS complex (when atrial depolarization occurs after ventricular depolarization), or no visible P wave (if atrial and ventricular depolarization occur at the same time). The third complex in this strip occurs early, therefore it is "premature," and is preceded by a negative P wave, leading to the assumption that it originated at or near the AV junction. It would also be appropriate, but less descriptive, to describe this complex as a premature atrial complex (PAC) as it originated in the atria.
3. The QRS complexes are narrow (<100 ms), no P waves are visible, the rhythm is regular, and the rate is slightly <100 bpm. This can be termed an accelerated junctional rhythm. The narrow QRS and lack of P waves imply that the rhythm is originating at or near the AV junction. If the rate was slow it might be called a junctional escape rhythm. If the rate was fast it would be a "junctional" (atrioventricular re-entrant) tachycardia. Since the rate is normal, the term accelerated junctional is appropriate.
4. The QRS complexes are narrow, the P waves are positive in lead II, one P wave is associated with each QRS complex in a consistent fashion, and the rate is slow (<60 bpm) and regular. The rhythm is sinus bradycardia.

Chapter 3

1. A lower case f is used to describe the baseline undulations (whether "coarse" or "fine") seen in atrial fibrillation, while an upper case F denotes the sharp, saw-toothed waves of atrial depolarization in atrial flutter.
2. In atrial flutter a pattern of F waves to QRS complexes is seen. For example, if every third F wave is followed by a QRS complex then atrial flutter with 3:1 conduction is said to be present. Sometimes with atrial flutter the AV conduction is

variable, for example it may vary between 2:1 and 3:1 conduction, but patterns still exist. With wandering atrial pacemaker (and multifocal atrial tachycardia) the rhythm is very irregular as depolarizations are initiating from several different areas of the atria. In atrial fibrillation the QRSs also appear very irregularly, however, in this rhythm no P waves are present.

3. Atrial flutter with variable AV conduction. The classic "saw-toothed" F waves of atrial flutter are apparent. The F waves are appearing at a rate of around 250 to 300 per minute, which is typical of flutter. At the beginning of the strip the atrial (F wave) rate is four times the ventricular (QRS) rate, indicating 4:1 conduction; toward the middle and end of the strip 3:1 and 2:1 conduction are seen.
4. Atrial fibrillation with a moderate ventricular response. The baseline shows undulations, but no regularly recurring organized atrial activity and the R-R intervals vary with no pattern indicating atrial fibrillation. The average ventricular rate is around 75 bpm, which would be considered a "moderate" rate.
5. This is *not* atrial fibrillation, but rather normal sinus rhythm. Although the baseline appears chaotic, careful inspection reveals a P wave in front of every QRS complex (not present in atrial fibrillation) and a regular ventricular rate (also not present in atrial fibrillation). The baseline deviations are actually artifactual, caused by patient movement.

Chapter 4

1. Normal sinus rhythm with frequent multiform PVCs. The underlying rhythm shows a P wave for each "narrow" QRS complex, with a consistent and normal relationship between the P waves and QRS complexes, normal rate, and regularity, therefore, this is normal sinus rhythm. The second, sixth, and eighth QRS complexes are early, "wide," appear different from the normal QRS complexes, and not preceded by P waves; they are PVCs. Two different PVC morphologies (in the same lead) would be sufficient to use the term "multiform"; in this case three different QRS morphologies are seen for the early beats.
2. Idioventricular escape rhythm. No P waves are seen, the QRS complexes are "wide," and the rate is very regular and slow. The individual QRS complexes may look like PVCs, but in this case are not premature. Rather this is a situation when the failure of the "higher" pacemakers (i.e., sinus and AV node/junction) allowed a slower ventricular pacemaker to take control of the rhythm.
3. Monomorphic ventricular tachycardia (possibly sustained). It is not possible to tell from this short strip, but if this tachycardia lasted more than 30 seconds it would be considered "sustained." Since all of the PVCs have essentially the same appearance, it is considered "monomorphic."
4. Nonsustained polymorphic ventricular tachycardia (torsade de pointes). This could be described as a "seven beat run of torsade" as it terminates after seven complexes. The "points" of the QRS twist upward (first four complexes) and

then downward, indicative of torsade de pointes. Certainly the PVCs do not all have a similar appearance, making it "polymorphic."

Chapter 5

1. Ventricular paced rhythm (normal sinus rhythm with pacing that is tracking the atrial rhythm). It is possible that this patient has a dual chamber pacemaker. The atrial rate shown would likely be above the threshold for pacing of the atria, so no evidence of the atrial component would appear on the ECG. It is also possible that this is a single chamber (ventricular) pacemaker.
2. Initially, this strip shows a ventricular paced rhythm (no P waves and pacemaker spikes followed by wide QRS complexes). P waves re-emerge prior to the third QRS complex, resulting in what may be a "fusion beat" as the depolarization initiated by the pacemaker spike and the depolarization initiated by the SA node may have merged, resulting in a sort of "dual" depolarization of the ventricles. Four beats of normal sinus rhythm next appear, and then the atrial rate slows, resulting in another probable fusion beat followed by the return of ventricular paced rhythm.
3. Accelerated junctional rhythm with intermittent failure to sense. The absence of P waves and a narrow QRS indicate that the rhythm is originating at the AV junction; the rate falls between 60 and 100 bpm, therefore, the term "accelerated" may be applied to indicate that it is faster than a typical junctional escape rate, yet too slow to be a "junctional" tachycardia. Pacemaker spikes appear following the QRS complexes (in the ST segments) of the second and sixth beats, indicating that the pacemaker sometimes fails to sense the native QRS complexes. These pacemaker spikes appear so soon after the QRS that the ventricles are likely still in a refractory period and are not depolarized again.

Chapter 6

1. Second-degree AV block Mobitz type I (Wenckebach). Note that the PR interval becomes longer with each successive P-QRS-T and then the third P wave is not followed by a QRS complex; the pattern of PR lengthening then restarts. This strip shows "grouped beating," which is always an indication to search for a Mobitz I.
2. Second-degree AV block Mobitz type II with 3:1 conduction. A constant pattern of three P waves followed by one QRS complex is seen, and the PR interval of the P waves that conduct is the same. This is not complete heart block as there is a relationship between the atrial and ventricular rhythms.
3. Initially it is normal sinus rhythm with a first-degree AV block. This is followed by a 2:1 second-degree AV block, which theoretically could be either Mobitz type I or Mobitz type II. The width and morphology of the QRS

complexes changes as well; this is not due to the AV block, but rather comes from a conduction defect (intermittent bundle branch block) discussed in Chapter 10.

Chapter 7

1. Electrodes are not leads. Electrodes are the conductive patches attached to the patient; leads are the electrical views obtained from the currents detected by the electrodes. The input from the right arm, left arm, and left leg electrodes (the right leg electrode simply functions as a ground) is manipulated in various ways in order to obtain six electrical "views" of the heart (leads I, II, III, aVR, aVL, aVF).
2. The initial event of depolarization of the ventricles usually is depolarization of the intraventricular septum, which proceeds from left to right. This relatively modest amount of current results in a small r in V_1 as the current is heading toward the positive pole of this lead. A small q is seen in V_6 as the current is traveling away from the positive pole of this lead. This is followed by the depolarization of the left and right ventricles; the net vector of this relatively large current is directed toward the left and thus toward the positive pole of V_6 (resulting in a large R wave) and away from the positive pole of V_1 (resulting in a large S wave).
3. The six leads—I, II, III, aVR, aVL, aVF—derived solely using the electrodes placed on the extremities are referred to as the limb leads, while the six leads—V_1, V_2, V_3, V_4, V_5, V_6—derived using individual electrodes placed on the chest are called the chest leads.

Chapter 8

1. Right axis deviation +120.* The QRS complex in lead I is more negative than positive, while that in Lead aVF is more positive than negative. This implies that the axis is in the right quadrant. Lead aVR has the most isoelectric QRS complex, so the axis is roughly perpendicular to this lead and therefore roughly +120 degrees.
2. Left axis deviation –30. The QRS complex in lead I is mainly positive, while that in lead aVF is mainly negative. This puts the axis in the left quadrant. Since lead II is the most isoelectric lead the axis is roughly perpendicular to this lead and therefore numerically closest to –30 degrees. The true axis must be slightly more negative than –30 as the QRS complex in lead II is slightly more negative

*The ECG in question 1 is from a patient with right bundle branch block, a conduction abnormality discussed in Chapter 10. This condition results in terminal rightward directed electrical forces due to delayed depolarization of the right ventricle. These terminal forces are unopposed by left ventricular activity. Under these conditions, debate exists as to how to properly determine axis. For the purposes of this quiz, we have used normal techniques for axis determination.

than positive and therefore directed slightly more toward the negative pole of lead II than the positive pole of lead II. This means that left axis deviation is present.

3. Normal axis +60. The QRS complex in leads I and aVF are both mostly positive, thus the axis is in the normal quadrant. The most isoelectric lead is aVL. Since the axis is roughly perpendicular to lead aVL and in the normal quadrant, it must be closest to +60 degrees.

Chapter 9

1. **(a)** Multiple signs of possible chamber enlargement are present: the P waves in lead II are both tall (RAA) and wide (LAA), the terminal negative component of the P wave in lead V_1 is "a box wide and a box deep" (LAA), the R waves in lead aVL are tall (LVH), and V_1 has deep S waves while V_6 has tall R waves (LVH). Although not the classic "strain" pattern, the ST segment depressions in the lateral precordial leads may represent repolarization abnormalities associated with hypertrophy.

   (b) The R waves in V_1 are taller than the S waves, but RBBB (Chapter 10) is present. Normally a "tall" R wave in V_1 is not considered evidence of right ventricular hypertrophy in the presence of RBBB because the tall R is an R' that is reflecting right ventricular forces unopposed by left ventricular forces (in RBBB the right ventricle continues to depolarize after the left ventricle has ceased); however, a "very tall" R' in this setting (sometimes defined as >15 mm) is evidence of possible RVH in the setting of RBBB. Although the "very tall" R in the ECG is suggestive of RVH, the T wave inversions in V_1 cannot be attributed to a "strain" pattern as they are likely associated with the RBBB.

   (c) Voltage for left ventricular hypertrophy is present as the R waves in aVL are taller than 12 mm; mild ST segment depressions are present in lead aVL but are not deep enough to represent a typical "strain" pattern.

Chapter 10

1. In both types of bundle branch block the slower cell-to-cell depolarization of a ventricle (left in the case of LBBB and right in the case of RBBB) results in a prolonged QRS duration (>100 ms). With RBBB the initial event of ventricular depolarization is normal septal activation; this current is directed toward the right (r in V_1, q in V_6), the right and left ventricles are activated next (S in V_1, R in V_6), and the terminal event is the continued depolarization of the right ventricle (R' in V_1, s in V_6). The overall pattern therefore is an rSR' in V_1 and a qRs in V_6. With LBBB the septum does not depolarize first, so the "septal r" of V_1 is lost as is the "septal q" of V_6. The initial event is activation of both ventricles with the net vector directed toward the left (Q in V_1 and R in V_6). The left ventricle continues to depolarize after the right ventricle has completed activation,

resulting in continued current flow toward the left and therefore a wide QS in V_1 and a wide (sometimes notched) R in V_6.

2. This term is used in cases where the QRS duration is prolonged, but the cardiogram shows neither a RBBB nor a LBBB pattern.
3. **(a)** Complete left bundle branch block. The QRS complex is >120 ms in duration, and the wide R (with no septal q) with T wave inversions and ST depression in V_5 and the QS with ST elevation in V_1 is typical of LBBB. The QRS complexes are preceded by normal looking P waves, and the PR intervals are consistent, thus (with one exception) the wide QRS complexes are not due to beats originating in the ventricles. The second QRS on the rhythm strip is early, wide, and bizarre (with a different morphology than the other QRS complexes, which are wide because of the LBBB), is not preceded by a P wave (but appears to have a P wave in the ST segment likely due to retrograde conduction from the ventricles to the atria), and is followed by a compensatory pause. This is a PVC.

   (b) Complete right bundle branch block. The P waves have a consistent relationship to the QRS complexes; therefore, the wide QRS complexes are not likely to be ventricular in origin. These wide QRS complexes (>120 ms in at least one of the limb leads) in conjunction with the RSR' in V_1 and the s wave in V_6 are consistent with RBBB.

Chapter 11

1. **(a)** Old inferior wall MI. Significant Q waves are seen in leads II, III, and aVF.

   (b) Evolving anteroseptal wall infarct, old inferolateral wall Infarct. The significant Q waves in leads II, III, aVF, I, and aVL are suggestive of an old inferolateral wall MI. The pattern of ST segment elevations with the beginnings of T wave inversions is suggestive of an evolving anteroseptal wall MI.

Chapter 12

1. Several things differentiate pericarditis and STEMI patterns. Perhaps the most obvious is that the ST segments and T waves typically go through a more drastic series of changes during a STEMI, while the pericarditis pattern is more subtle and stable. With STEMI initially (and often gone by the time the patient arrives at the hospital) T waves are tall and peaked ("hyperacute T waves"), this is followed by ST segment elevations (often accompanied by new Q waves), which later come toward baseline as the T waves invert. If Q waves have not previously appeared they typically appear in this phase. In the long run the ST segments of a STEMI patient may return all the way to baseline or remain modestly elevated, while the Q waves typically remain. In contrast, tall T waves are not seen with pericarditis, the ST segment elevations are more modest in amplitude and change much more slowly, and Q waves do not

develop. Other differences include: pericarditis is more global usually causing ST elevations in all leads except AVR and PR segment depression is often seen with pericarditis, but not with STEMI.

2. Both STEMI and early repolarization are associated with ST segment elevation. As described in the answer for question 1, the ST segments go through a series of changes during a STEMI. ST segment elevations associated with early repolarization are stable. New Q waves typically result from STEMI, but not from early repolarization. The ST segments of a STEMI typically have a convex ("frown" or "tombstone") shape, while those of early repolarization have a concave ("smile") appearance. Finally, the J point (where the QRS complex ends and the ST segment begins) often has a short little upswing ("hitch") with repolarization; this is not typical of STEMI.
3. The "triad" of WPW consists of (a) wide QRS complex, (b) short PR interval, and (c) delta wave. The first of these is seen with most conduction defects (RBBB, LBBB, IVCD), the latter two are not. Hemiblocks (also conduction defects) alter the QRS axis but do not shorten the PR interval or cause delta waves and typically have normal QRS durations.

Chapter 13

1. Normal sinus rhythm. The QRS complexes are wide because of the RBBB (rSR' in V_1, S in V_5); lead II shows upright P waves with normal, consistent relationships to the QRS complexes demonstrating that this is not a ventricular rhythm.
2. Ventricular bigeminy. Every second beat is a PVC.
3. Atrioventricular nodal re-entrant tachycardia. The rate is fast and very regular and P waves are either absent or possibly negative P waves are occurring after the QRS complexes.
4. Accelerated junctional rhythm (ectopic atrial rhythm). Negative P waves are present before each QRS, indicating a junctional (also known as ectopic atrial) rhythm. The rate is between 60 and 100 bpm, so the term accelerated is often used (slower junctional rhythms are termed "escape").
5. Atrial flutter with variable AV conduction. Saw-tooth F waves are apparent, and the ratio of F waves to QRS complexes varies.
6. Normal sinus rhythm with complete (third-degree) atrioventricular block and an idioventricular escape rhythm. Normal appearing P waves are regularly occurring (some of the P waves are falling on T waves or in ST segments) at a normal rate, implying the atria are in normal sinus rhythm. Wide QRS complexes are occurring regularly at a very slow rate, implying origination in the ventricles. The two rhythms (atrial and ventricular) appear independent of each other.
7. Torsade de pointes. Initially sinus rhythm with complete heart block appears to be present, then a short run of PVCs that are not all alike in appearance occurs. By definition this is a (nonsustained) polymorphic ventricular tachycardia. As the "points" of the QRS complexes of the PVCs first point down and then point up, this seems to be torsade de pointes.

8. Accelerated junctional rhythm followed by normal sinus rhythm. Initially the narrow QRS complexes with no associated P waves imply a junctional rhythm, then upright P waves appear, suggesting that the sinus node has recaptured the rhythm.
9. Accelerated junctional rhythm with a PVC followed by an accelerated junctional rhythm from a different ectopic focus. Negative P waves with a narrow QRS imply a junctional rhythm, a "wide and bizarre" QRS (PVC) intervenes, and when the junctional rhythm resumes the QRS complexes are still narrow, but P waves are not seen and the rate is different, implying that a different part of the "junction" may have taken over the rhythm.
10. Second-degree AV block Mobitz type I (Wenckebach). The PR intervals lengthen progressively, then a P wave is not followed by a QRS complex.
11. Monomorphic ventricular tachycardia. A continuous string of PVCs with a consistent appearance; if this lasts for more than 30 seconds it would be sustained ventricular tachycardia.
12. Ventricular fibrillation. No regularly occurring organized ventricular electrical activity is seen, but rather chaotic fluctuations of the baseline. The fluctuations are relatively large so this would often be called a "coarse" ventricular fibrillation. The patient would be unresponsive with no pulse or blood pressure with this rhythm.

Chapter 14

1. Although the baseline is wandering the ST segments are consistently depressed and meet the criteria of three of more complexes in a row showing significant depression.
2. (a) 1 mm of down-sloping ST depression
   (b) 0.5 mm of horizontal ST depression (not significant)
   (c) 2 mm of down-sloping ST depression
   (d) T wave inversion (ST segment is isoelectric)
   (e) 2 mm of down-sloping ST depression
   (f) isoelectric

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